market, some are delivered, some are constructed on site. Again, taking an animal's response, it is not important whether they are made from timber, steel or plastic. Of course the expense of maintenance would vary between different types of material used but the most important aspect is whether the partition will allow animals to share space between adjacent partitions. Sharing space between partitions will ensure that cows are allowed sufficient space to swing the head and transfer the centre of gravity forward to allow them to get up comfortably.

The extent of the "space envelope" for the rising movement is equivalent to the cow's body length plus some 0.7 - 1.2 m. As it would be uneconomical to provide so much space over the cubicle bed and to try to keep the cubicle bed free from soiling it is important to provide "space sharing" cubicle partitions. Such partitions allow cows to insert their heads through the partition and to move forward to get up easily. Some examples of "space sharing" partitions for mature 700 kg dairy cows are shown in the accompanying slides.

Cubicle partitions should be set out 1.2 m apart. This would allow large framed 750 kg cows, in calf, sufficient room to lie down. Any division rails should be set at 400 mm (say 16 inches) to accommodate the curvature of the rib cage without causing unnecessary bruising.

Cows are indiscriminate defectors; they will defecate while lying down or standing up. In order to minimise soiling of cubicle beds cubicles should be fitted with headrails

placed on top of the cubicle partition, some ¼ of the length of the cubicle bed. The rail should be set some 250 mm below the withers so as to position the cow's rectum close to the edge of the cubicle bed, this should help to keep the bed clean.

In some layouts cubicles are arranged to face each other (head to head). Any division barrier which has been installed to prevent cows entering the opposite cubicle should not be higher than 225 mm above the bed. This will allow the cow to lounge forward without hindrance. On the assumption that there is a solid wall in front of the cubicle, it is important to ensure that cubicle partitions are designed to allow cows to "borrow" space, so that they can swing their heads and necks into a neighbouring cubicle.

The British Code of Recommendations for the Welfare of Livestock (Cattle) (2) calls for the provision of a dry lying area. Well fed dairy cows, like other ruminants, are very tolerant of low temperatures but would still seek sites of enhanced comfort ie dry, resilient surfaces, away from draughts. Such sites tend to be subject to competition and tend to get taken before other locations.

On firm levelled surfaces, bedded with straw, cows will spend some 14 hours a day lying down as opposed to only 7.5 hours a day on bare concrete. Free choice of access experiments showed that rubber mats can also improve animal comfort but cows spend about 3.5 hours a day less lying down on these mats compared with the straw.

Cows have evolved on pastures, away from slurry and hence from the veterinarian standpoint the less the cows' hooves are exposed to slurry the better it is. The cow's hoof horn will remain harder and would be more resistant to abrasion if kept dry. It is important therefore to remove slurry frequently from cubicle passages, collecting yards and other areas. This will also reduce the lubricating effect of slurry and reduce skidding and possible falls and the subsequent upper leg lameness.

Slippery concrete floors are perennial problems in both new and old dairy units. It is recommended that newly laid concrete floors are finished with a wooden float so as to generate a high grip, non-skid floor surface. Experimental work, based on a precise computer controlled "foot test rig" showed that optimum slip-resistant floors for mature cattle should have a pattern of grooves spaced at 40 mm intervals. In sites encouraging a high volume of animal traffic, eg feeding areas etc, a pattern of hexagons with 46 mm sides formed by 10 mm grooves would provide improved anti-slip characteristics(3).

Conclusions

Many factors often acting together are thought to be responsible for the increased incident of lameness in the herd of dairy cows. Poor housing, in terms of broken, slippery, slurry covered concrete floors, small and ill designed cubicle partitions and mangers can be responsible for damage to the legs and hooves and thus lead to lameness problems.

Building designers and veterinarians can advise and influence producers and look critically and in detail at their housing systems and try to look for those relatively small improvements in the design of cubicles, floors and mangers which can contribute to the improved health of the herd.

The provision of "space-sharing" cubicles, well bedded, dry and resilient lying areas, non-slip floors and animal friendly shapes of mangers can go a long way towards keeping animals more comfortable, healthier, safer and "happier".

References

1. Potter M J and Broom D M, Behaviour and welfare aspects of cattle lameness in relation to building design p 80 - 84. Update in cattle lameness. Proceedings on the VIth International Symposium on Diseases of the Ruminant Digit. The British Cattle Veterinary Association, July 1980.

2. Codes of Recommendations for the Welfare of Livestock (Cattle). MAFF Publications PB0074.

3. Dumelow J and Allbutt R, The effect of floor design on skid resistance in dairy cattle buildings. p 130 - 142. Update in cattle lameness. Proceedings on the VIth International Symposium on Diseases of the Ruminant Digit. The British Cattle Veterinary Association, July 1980.

ENVIRONMENTAL CONTROL OF LAMINITIS IN DAIRY COWS

L.C. Allenstein

306 Pleasant St.
Whitewater, Wisconsin, 53190
U.S.A.

It is well known that the major control measurer for laminitis in dairy cattle concerns the ration being fed. However, many management and environmental factors contribute to the degree of laminitis and to its reoccurrence or extension of the disease. After correcting the forage to concentrate ratio, the level of A.I.F. and NIF, the amount of carbohydrate, and the fineness of the ration mix, one must also look at the: 1) delivery of the ration, 2) the availability of the ration (bunk space), 3) trauma to the feet due to cement, 4) the stall surface, 5) exercise provided, 6) resting time allowed, 7) the management of raising heifers and dry cows, 8) study genetics of your cattle, 9) procedure followed in "steaming up" the heifers and dry cows. Monitoring these factors will definitely aid in keeping laminitis under control. This short paper will consider yard and stall surfaces on which cattle must stand.

FUNCTIONAL TRIMMING

E. Toussaint Raven
Groenekanseweg 110
3737 AJ Groenekan
the Netherlands

'Functional trimming' is about the basic care that can be given to cows that are victim to man-made claw diseases. We altered in many parts of the world, depending on so many circumstances, the conditions of life of our dairy cattle that much away from a normal ruminant life, that the physiological process in the organism was affected.
One organ in the cow's body appears to be very tender to this intervention, namely the corium of the claw (or pododerm). One reason for this is its loaded and pinched (between claw bone and horny shoe) position under the bodyweight in the horny capsule, prone to contusion.
All this is no new information, but it leads to the conclusion (that also is not new, but worth to be repeated according to the opinion of this congress' organisation) that problems due to pododermatitis (90% of cattle lameness?) are not exception, but rule in intensive dairy farming. A man-made, multifactorial problem in the dairy cow, in the control of which functional trimming can play an important part.

What happens, in seriousness depending on the quality of so many circumstances (not at least the resistance of the animal):
Stressed physiology (nutrition = *laminitis*) and uncontrolled bacteriology (housing = *heelhorn erosion*) lead to unsuitable hornformation in the claw. This leads to pressure problems (= intracompartmental pressure) on the corium due to inefficient weight distribution (= contusion -

ENVIRONMENTAL CONTROL OF LAMINITIS IN DAIRY COWS

L.C. Allenstein

306 Pleasant St.
Whitewater, Wisconsin, 53190
U.S.A.

It is well known that the major control measurer for laminitis in dairy cattle concerns the ration being fed. However, many management and environmental factors contribute to the degree of laminitis and to its reoccurrence or extension of the disease. After correcting the forage to concentrate ratio, the level of A.I.F. and NIF, the amount of carbohydrate, and the fineness of the ration mix, one must also look at the: 1) delivery of the ration, 2) the availability of the ration (bunk space), 3) trauma to the feet due to cement, 4) the stall surface, 5) exercise provided, 6) resting time allowed, 7) the management of raising heifers and dry cows, 8) study genetics of your cattle, 9) procedure followed in "steaming up" the heifers and dry cows. Monitoring these factors will definitely aid in keeping laminitis under control. This short paper will consider yard and stall surfaces on which cattle must stand.

FUNCTIONAL TRIMMING

E. Toussaint Raven
Groenekanseweg 110
3737 AJ Groenekan
the Netherlands

'Functional trimming' is about the basic care that can be given to cows that are victim to man-made claw diseases. We altered in many parts of the world, depending on so many circumstances, the conditions of life of our dairy cattle that much away from a normal ruminant life, that the physiological process in the organism was affected.
One organ in the cow's body appears to be very tender to this intervention, namely the corium of the claw (or pododerm). One reason for this is its loaded and pinched (between claw bone and horny shoe) position under the bodyweight in the horny capsule, prone to contusion.
All this is no new information, but it leads to the conclusion (that also is not new, but worth to be repeated according to the opinion of this congress' organisation) that problems due to pododermatitis (90% of cattle lameness?) are not exception, but rule in intensive dairy farming. A man-made, multifactorial problem in the dairy cow, in the control of which functional trimming can play an important part.

What happens, in seriousness depending on the quality of so many circumstances (not at least the resistance of the animal):
Stressed physiology (nutrition = *laminitis*) and uncontrolled bacteriology (housing = *heelhorn erosion*) lead to unsuitable hornformation in the claw. This leads to pressure problems (= intracompartmental pressure) on the corium due to inefficient weight distribution (= contusion -

swelling) and circulatory disturbance (= swelling). Contusion and swelling in a narrow and pinched situation lead easily to ischemia, necrosis and ulceration, ... and pain. This is lameness as a herd problem in our dairy cattle. (Digital dermatitis or Mortellaro disease can be a herd problem as well, but cannot be controlled by trimming).
This contusion-conception is build on observation and clinical consideration. As a consequence *functional trimming* was developed, the result of which supports the validity of the theory. Statistical evidence for the correctness was not searched for by the author because of the difficulty, if not impossibility, to find comparable material (too multifactorial origin, the factor 'farmer' being the most unsafe).

So far an introduction to functional trimming as part of foot care in general. In fact, accepted the animals and the breed, foot care is based on three pillars: housing (= farmer), nutrition (= farmer) and periodical trimming. Trimming because the unfavourable effects of housing and nutrition on hornformation are difficult to eliminate with our present knowledge and our want for milk. Even if housing and nutrition satisfy to the best possible conditions, unsuitable hornformation will still exist to a certain extend. At need, this can be controlled by functional trimming; that is a way of trimming based on the knowledge of the pathogenesis of the easily necrotising inflammations of the corium.
We shall discuss with some slides the influence of heelhorn erosion and laminitis (the two, multifactorial diseases of the

hornproducing tissues) on the loading and the pressure, on the corium between claw bone and horny shoe, and the method of trimming as a logic consequence of these findings.
As for the results: they are good. Today lame, tomorrow no more lame is clear and needs no statistical support.

Veterinary literature accepts already for a hundred years that claw trimming is important in the prevention and treatment of claw-lame cows. Rules however were never given and the farmer (cow-servant) was the person supposed to do it.
Today our experience learned us: Strict rules are needed to become a satisfying result, and the work should be done (apart from exceptions) by professionals.
An attendant circumstance is the fact that functional trimming (or preventive trimming) and curative trimming (= the treatment of choice in claw-lame cows) are the same skill. This means that well-educated professional claw trimmers can treat claw-lame cows. It also means, in my opinion, that veterinarians have to learn functional trimming to be able to treat claw-lame cows.

Learning functional trimming needs supervised training: course organisation - teachers. Such a course prepares claw trimmers to do 90% of herd prevention (preventive health control through periodical trimming to limit the vulnerable effect of unsuitable hornformation) and veterinarians to do 90% of treatment of individual lame cows (where prevention failed or the problem was not recognized in time). For both professions there is work enough; they should not work in competition but in cooperation. The two professions are complimentary to one another in health care.

Health care in foot problems starts with farmer's education. He, or she is the

person that makes the problem. She/he should be the first to recognize it and ask for help. But where is the veterinarian and where is the claw trimmer to do a good job? There is a specialised training centre in Holland , but that is far away. Canada, if it feels the need, should build its own.

Conclusion:
If you survive my slides and if you get interested, the benefit of the doubt should give you the enthusiasm to read, carefully, Cattle Footcare and Clawtrimming. If you are still enthusiast you need your course; for nobody can learn a skill without supervised training, and without the skill you cannot make results to judge.

BANFF, June 1994

GENERAL MANAGEMENT OF CLAW LAMENESS.

Alberto Brizzi
via Gorizia n. 16 43100 PARMA Italy

Introduction

Modern dairy farming is an intensive activity where few operators care for large numbers of animals.

Time available for the care of a single diseased cow frequently shrinks to a point that culling is considered a profitable option against prolonged individual care.

How do we deal with this situation when treating a lame cow ?

To keep trouble for the herdsman to a minimum, and knowing that cows separated from their groups can be neglected and/or udergo deep changes in the ration they are fed, *we must try to avoid the separation of the patient from the herd.*

To reach this goal we have to restore, as soon as possible, a decent locomotor function, since this furthermore reduces, sometimes greatly, the economical impact of lameness.

Foot care is a labour intensive activity, it is therefore important to keep the necessity of subsequent interventions on the same case to a minimum.

The basic idea is: *do whatever necessary on first intervention and limit further need for individual care to those animals who do not respond at first hand".*

The "ambulating" treatment of lameness.

Recovery from lameness of a cow, while she walks with the herd, is possible if:

a) we establish an accurate prognosis on first examination and discuss the implications with the owner/manager;

b) we examine (and trim) all feet of the patient. There can be more than one foot affected and we don't know unless we lift all four; possibly this will relieve and/or prevent problems to other feet.

c) we carefully perform functional trimming trying to achieve maximum rest for the diseased claw(s). General

guidelines for therapeutic trimming, as set by Drs. Toussaint Raven, are well known:

1) Give the sound claw stability.
2) Unload the diseased claw by lowering it at the heel.
3) Thin out any hard edges of the horn which cause undue and harmful compression on the quick around open defects of the claw.
4) Apply an orthopedic sole, under the omolateral sound claw, as soon as the lesion appears complicated or whenever it is evident that loading the affected digit would lead to eccessive locomotor impairment.

d) we cause no or very limited haemorrage, and therefore can avoid bandaging. Bandages need control and renovation, time consuming work, and, first of all, a *dry environment*, which is particularly difficult to achieve in a modern free stall barn without separating the patient from the herd. The protection eventually needed by the diseased digital structures can be provided by the application of an orthopedic sole, which furthermore allows total unloading of the diseased claw.

Parenteral administration of antibiotics is used to control eventual septic complications affecting the deep digital structures.

The results of this approach are presented in the following paper: "Claw trimming and claw lesions".

Collateral preventive measures.

Sound management practices will reduce the occurrence of digital lesions.

Correct feeding.

The farmer should be aware that laminitis, in its various forms, plays an important role in the incidence of some digital lesions (sole ulcers, white zone lesions).

Correct feeding practices are capable to reduce greatly the impact of laminitis; we can't discuss the item here, but particular attention should be paid to the quality of the feedstuffs used.

It should also be remembered that the transition from dry period to lactation, together with the first three/four months of lactation can be problematic, particularly in heifers.

Any evidence of feeding problems should lead immediately to research for the causes and the appropriate corrective measures.

It should be remembered that claw problems due to improper feeding occur generally after a lag period of about two months.

Environmental Hygiene.

Foot hygiene is often neglected, the sight of cows walking 10 cm deep in the "slurry" is so common that we look at it as to something normal.

Cleanliness and dryness of the floors are of extreme importance in the prevention of those diseases which have a recognized infectious ethiology, such a interdigital and digital dermatitis.

Desinfection of the claws can effectively be accomplished with the use of walk-through formalin baths.

Provided the amount of solution and the formalin concentration used are sufficient, footbathing prevents, under Italian circumstances, the recurrence of digital dermatitis and inhibits heel horn erosion.

Formalin footbathing is contraindicated in case of open wounds in the claws or in the interdigital space, because formalin fixates living tissues and inhibits healing processes.

Digital dermatitis control requires a coordination of different measures if success is to be attained.

The first form of prevention is treatment of the clinically active lesions, as these apparently are a source of infection. Topical treatment of digital dermatitis lesions, with an oxytetracycline based spray according to Cornelisse et al. [1], is a common practice in Europe and has not lead to recognizable residues in milk, nor in blood of treated animals [2] with a detection limit of 5 ng/ml. Topical treatment should be performed, within the shortest time, on all clinically evident lesions, ideally as part of a generalized foot-trimming program.

Regular footbathing with 3-5% formalin solutions must follow individual treatment. This is normally done in walk-through baths which are located at the exit of the milking parlour to limit the risk of milk contamination.

Some serious attempts to modify the environmental conditions of the barn should be made, particularly to prevent puddle formation in the alleys.

Conclusions

Control of lameness in cattle is difficult, if durable results are to be achieved on a short term basis, trimming and treatment of lame animals are fundamental, to maintain the gained results many other measures must be discussed and taken together with farmer, nutritionist and veterinarian.

References

1 Cornelisse J.L. Peterse D.J. Toussaint Raven E. (1981) - Een nieuwe aandoening aan de ondervoet van het rund. Dermatitis digitalis ? - Tijdschr. Diergeneesk. *106*, 452 - 455.

2 Anfossi P. Roncada P. Tomasi L. Brizzi A. Stracciari G.L. (1993) [Oxytetracycline determination in serum and milk of cows topically treated with a spray formulation] Atti SIB XXV, 431 - 435.

CLAW TRIMMING AND CLAW LESIONS

Alberto Brizzi
via Gorizia n. 16 43100 PARMA Italy

Introduction

That some relation exists between claw trimming and claw lesions is a well known fact, it appears however difficult to quantify the advantages of claw trimming in a herd.

The main reasons for this lie in the multifactorial nature of digital diseases and in the fact that foot care can be performed in many different ways with consequently different results.

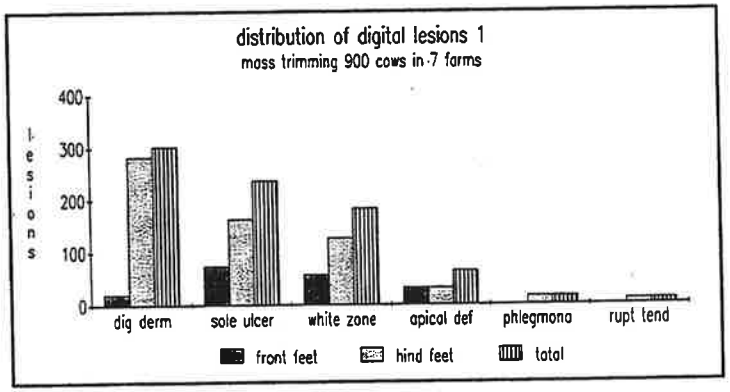
The results presented in this paper were obtained with *functional trimming* as it was developed by Drs. Toussaint Raven and is currently taught at the Practical Training Centre for Cattle Husbandry and Grassland Management at Oenkerk (The Netherlands).

Claw trimming should be learnt under the supervision of a qualified instructor.

Distribution of digital lesions in cattle.

The following two graphs show the distribution of the most frequent foot lesions among the cows of several dairies located in the Po plane.

A foot may suffer from more than one lesion, therefore the total amount of recorded lesions has no



strict relation with the number of feet examined.

The first graph shows the distribution of the principal lesions observed, during massive trimming, in 7 herds (about 900 cows).

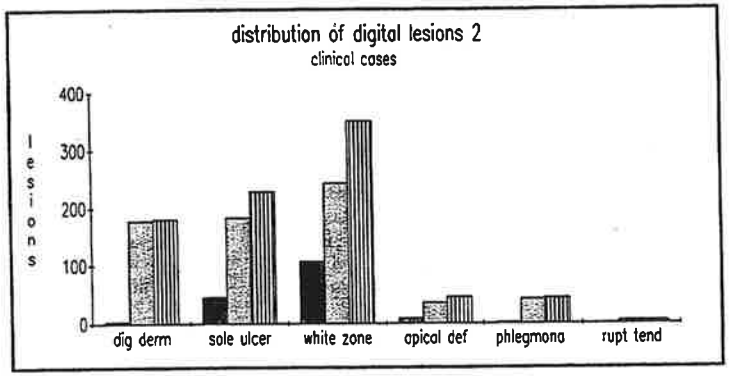
The most frequent disease appears to be digital dermatitis (dig. derm.), followed by *pododermatitis circumscripta* (sole ulcer) and white zone disease (white zone) with the correlated underminings of the wall.

A rather large separate class (apical def) comprehends the lesions of the anterior margin of the claw. These lesions usually occur as a complication of digital dermatitis. A typical lesion undermines the anterior wall, causing a permanent vertical fissure in the horn, this syndrome is often accompanied by an apparently progressive osteolysis of the third phalanx.

Pain at the toe forces the cow to walk on the heels of the affected foot, the alteration of the gait is evident and the hyperextension of the digit does not allow the use of an orthopedic sole. The affected animals often maintain a fair body condition and acceptable milk yield.

Interdigital phlegmon (phlegmona) and its sequelae are quite rare, as mass trimming findings, since they are normally treated, as soon as they occur, with the administration of antimicrobial drugs.

The rupture of the deep flexor tendon (rupt tend) is a permanent lesion, mostly caused by the



complication of a sole ulcer, its occurrence appears marginal.

The second graph shows the distribution of foot lesions in animals which were brought to the claw trimmer's attention because of locomotor distress.

It appears clear that white zone lesions are clinically more evident than those due to pododermatitis circumscripta and digital dermatitis which are less apparent, particularly if bilateral.

Claw trimming as a therapy.

Claw trimming is effectively used to treat lesions of the horn forming structures.

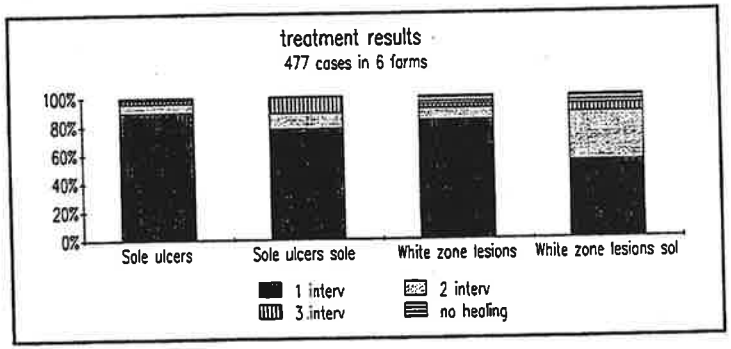
As a rule the tissues underlying the horn layer are left, as far as possible, untouched and no bandages are applied.

When complications occur, like the inflammation of deep digital structures, collateral measures, like the application of an orthopedic sole and/or the parenteral administration of antimicrobial drugs, are used.

To try to describe the effectiveness of this kind of approach the lesions were subdivided in two classes, depending on the subjective need for the application of an orthopedic sole (sole ulcer / sole ulcer sole).

A gross evaluation of treatment results is possible looking at the number of interventions needed for the recovery of the patient.

The following graph shows the percentages of recoveries after different numbers of interventions in



477 lesions due to *pododermatitis circumscripta* and white zone disease in 6 different herds.

The lesions classified as "no healing" should be considered as chronic or unhealable with the use of simple claw trimming.

Pododermatitis circumscripta (sole ulcers) has the best outcome, if an orthopedic sole is used at the appropriate time, complications are rare.

The situation is different with white zone lesions, the prognosis is worse especially in those cases when undermining of the wall has occurred.

Among those cases which required an orthopedic sole, the percentage of "no healing" lesions lies somewhere around 6%.

The effectiveness of claw trimming as a therapy of horn defects depends on many factors which are: 1) localization of the lesion; 2) conformation of the claw; 3) presence of laminits; 4) housing; 5) milk production and stage of lactation; 6) timing of treatment; 7) age of the animal and 8) sex of the animal.

1) Localization of the lesion.

As we move from the heel towards the toe of the claw, the prognosis of the encountered lesions becomes more and more serious.

The less dangerous lesions are normally located at the heel or at the back of the sole, lesions of the wall are more difficult to treat and often require an orthopedic sole.

Lesions located at the toe, when combined with overconsumption of the claw, or with a lesion of the coronary band, are normally serious and can prove unhealable.

2) Conformation of the claw.

In Italian cattle corkscrew claw deformities, in inner front claws and in both inner and outer hind claws, are increasingly frequent.

The diffusion of this condition has brought an increase in front feet lamenesses and the consequent need to provide those feet with regular trimming.

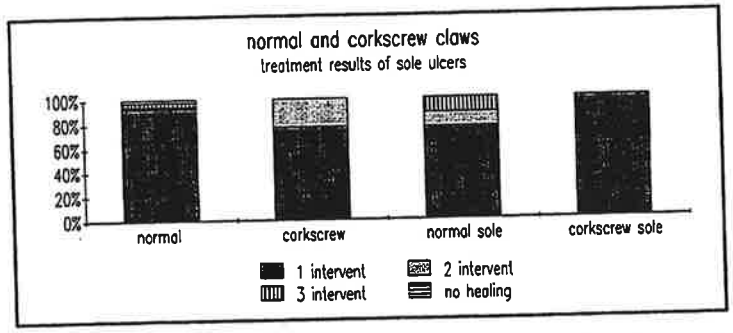
The inner conformation of corkscrew claws requires careful trimming in order to avoid accidental damage of the quick at the toe.

The altered direction of the claw structures, particularly of the abaxial wall, predisposes corkscrew claws to chronic laminitis and overgrowth.

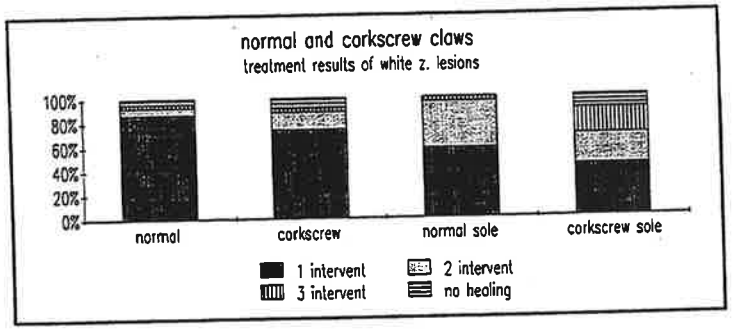
This fact is accompanied by a high frequence of white zone lesions, with possible undermining of the abaxial wall, which can prove particularly difficult to treat.

The following graph shows the results of treatment of *pododermatits circumscripta* lesions in normal and corkscrew claws (189 total cases).

The particular situation of corkscrew claws appears to affect only marginally the healing of *pododermatitis circumscripta*, if an orthopedic sole is used results are good.



Next graph shows the results of treatment of white zone lesions in normal and corkscrew claws (292 total cases).



White zone lesions affecting the wall appear difficult to treat in corkscrew claws; a considerable percentage of cases requires more interventions and even the use of an ortohpedic sole leaves a 10,7% of "no healing" lesions.

Since the most common horn defects respond less favourably to treatment when occurring in corkscrew claws, it appears evident that this conformation is undesirable and its occurrence should be reduced.

3) Presence of laminitis.

For the clawtrimmer, the most preminent feature of chronic laminitis is the presence of haemorragic discolorations in the horn of the sole and the white zone. These alterations are so frequent among dairy cows that, unless we pay special attention to them, they are easily overlooked and our attention is often caught by a sole which is not exhibiting any!

The disease determines an initial tendency towards horn overgrowth, which is particularly evident in young animals when housed on soft bedding. Old cows with chronic laminitis suffer, on the other hand, from increasing difficulties in horn production.

The degenerative vascular changes, determined in the corium by the disease, explain the problems we experience when treating horn defects in laminitic animals.

Any open horn lesion, if accompanied by evidence of laminitis, calls for the application of an orthopedic sole and should be accompanied by the emission of a guarded prognosis.

4) Housing

The quality of pavements, the environmental humidity, and the suitability of bedding material play a determinant role in the diffusion of digital lesions and have a strong influence on the results of therapeutic and preventive trimming.

When housing conditions are difficult, separation of the lame animal from its group should be seriously considered as an essential part of the treatment.

5) Milk production and stage of lactation.

High milk production is often correlated with laminitis and is accompanied by drainage of precious blood-carried nutrients operated by the mammary gland.

Horns of older cows show clear sings of each lactation in the form of growth rings. A similar reduction in horn production rate occurs in the claw too, predisposes fresh cows to overconsumption and weakens the claw's resistance to disease.

This is particularly true for heifers, whose smaller size accounts for a lower dry matter intake capability.

6) Timing of treatment.

Prompt treatment of uncomplicated horn defects is important but not absolutely necessary, most lesions do wait for some days without significant worsening of the prognosis. The economic loss for the farmer obviously increases as time passes but, as a rule, I do not encourage "do it yourself" emergency treatment of claw lesions by untrained owners or herdsmen.

In my experience complications of claw lesions occur more frequently as a consequence of improper aggressive interventions performed by unqualified people.

7) Age of the animal.

The age of the animal is correlated with difficulties in healing open defects of the claw, partly because of the frequent presence of chronic laminitis.

Older cows, housed the year round in tied stall barns, can have such a slow rate of horn formation that they normally are unable to adapt to free stalls with concrete floors. Remargination of horn defects implies horn formation: the slower the horn production, the slower the healing process.

8) Sex of the animal.

No direct relation between body size and claw dimensions is evident in cattle, mature bulls show often underdimensioned claws.

Relatively little claws in bulls explain why small losses of horn, which affect the bearing surface of the claw, like the ones seen in the interdigital dermatitis/heel erosion complex, can easily determine discomfort or even lameness in mature bulls.

Horn formation rate in males is slower than in females and, as as age progresses, this rate further slows down. Ageing heavy bulls must be trimmed

carefully in order to avoid overtrimming which can have dreadful consequencies.

Bulls of various breeds show marked asimmetry between outer an inner hind claws, with some kind of hypotrophy of the inner one. This conformation, accompanied by an increasing frequence of corkscrew claw deformities, is unfavourable to functional trimming and makes treatment of outer claw defects difficult. Glued-on orthopedic soles can however readily be used and often even allow normal semen collection.

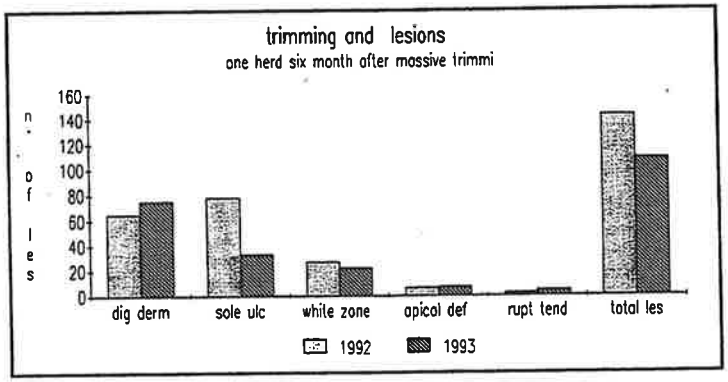
Claw trimming and prevention

The effect of claw trimming as a preventive measure varies. Results can be excellent (2 - 3 lame cows in six months after one mass triming session) or appear so bad that, in some farms, routine claw trimming appears questionable. Bad results are more frequent in herds where chronic laminitis occurs massively and where housing and managerial deficiencies are present.

The following graph shows the variation in the occurrence of foot lesions six months after the first massive trimming in a herd of about 215 cows.

The herd was characterized by a high prevalence of chronic laminitis and no footbathing facilities were available.

An overall reduction of about 20% in the number



of recorded lesions was evident and changes were seen in the occurrence of the single lesions.

The biggest reduction was seen in the number of sole ulcers, the effect on white zone lesions was less impressive.

Chronic lesions (like ruptured deep flexor tendons and anterior defects of the wall) increased slightly. Since these are permanent lesions, the slightest incidence increases the prevalence, unless the affected animals are culled.

A different consideration should be made for digital dermatitis which, being an infectious disease, is only slightly influenced by treatment and tends to increase unless effective sanitation measures, like formalin footbathing, are taken.

Conclusions.

There are so many differences among farmers, herds and farms that it can be really difficult to point out where the origin of a lameness problem lies; however claw trimming "never fails to give relief" and remains a fundamental control measure.

Durable or permanent reduction of lameness occurrence in a herd implies the adoption of many collateral measures on various fronts, including improvement of feed quality and feeding practices, genetics, housing, sanitation of the environment and general management.

References

Greenhough P.R., Mc Callum F.J., Weaver A.D. - Lameness in Cattle - 2nd ed. Wright Scientechnica, Bristol 1981.

Toussaint Raven E. - Cattle Foot Care and Claw trimming - Farming Press, Ipswitch 1985.

NUTRITIONAL FACTORS ASSOCIATED WITH LAMENESS

W. Robert Ward,
Liverpool University, Leahurst, Neston, South Wirral, L64 7TE, UK

Introduction

Feeding is one of several factors that can cause foot lameness in dairy cattle. Other important factors are genetic, physical (buildings, bedding, gateways), and human (farmers and foot-trimmers). Opinions range from blaming feeding as the major factor to suggesting that it is less important than other factors (David, 1990)

Nutrition is a subject on which practising veterinarians have mixed feelings. Most underwent a detailed undergraduate course, but many feel unable to feed a cow at the end. Nutritionists use computers or seem to pluck figures from the air, so that some veterinarians feel inferior.

So what is the role of the veterinarian in advising on a lameness problem with a possible nutritional component?

Types of lameness.

First, veterinarians can diagnose the type of lesion. An investigation, funded by the UK Ministry of Agriculture, looked at the amount of lameness in over 4,000 cows over two winters on 37 dairy farms in England and Wales from 1989 to 1991 (Clarkson and others, 1993).
Solar ulcer (Pododermatitis circumscripta) and White line abscess (White zone abscess) together accounted for over half the lameness. Both are believed to arise in part from failure of the corium to produce strong horn, and nutritional factors could well be one factor in this.
Acute laminitis accounted for only 2% of the lesions. This is often associated with either toxaemia, or with cereal overeating and rumen acidosis. 'Sub-acute laminitis' is a term commonly used for a lesion in the corium, but the sole has no laminae, and the lesion is perhaps not an inflammation. We did, however, recognise local haemorrhage or bruising in the ulcer site, and regarded that as an early stage in sole ulcer.

Foreign body penetration elsewhere in the foot (Traumatic pododermatitis) caused 5% of the lesions. This is often blamed simply on the foreign body, but soft or thin horn must sometimes be part of the cause, and that could have a nutritional component.

Skin lesions, such as interdigital necrobacillosis (foul) (%5 of lesions) and digital dermatitis (8%) are clearly caused by

micro-organisms, but the feeding system can influence the environment by producing a lot of liquid slurry.

Epidemiology.

Having diagnosed the lesion, the veterinarian can look at the number of cases of each type, and their distribution by season, stage of production, and type of animal.

Seasonal effects can be obvious, eg foreign body penetration associated with walking along a poor track in spring, when the feet are soft, or an outbreak of foul associated with feeding kale. Effects of winter housing and feeding on the corium, however, may not show until perhaps two or three months later, and a damaged corium may show a lesion indefinitely.

First lactation animals may suffer acute laminitis, and later sole ulcer, or white line abscess, because they are unused to cubicles and self-feed silage faces, and come onto concrete with feet soft from pasture.

Older cows may develop ulcers because of progressive increase in thickness of the outer hind claw, uncorrected by trimming and leading to extra weight-bearing. They may be larger cows, and may find it difficult to lie in small cubicles. Singh and others (1992, 1993b) observed that some cows lay for only 4 hours a day, while others lay for 12 hours, in a herd with silage fed in front of the cubicles. Sometimes one cow pushed another out of a cubicle in order to reach the silage.

An increase of lameness in one winter may be blamed on a change in silage. Unpalatable silage with low fibre content will lead to a big drop in the amount of fibre consumed and thus less saliva, so reducing the buffering of acid produced from fermentation of starch, and of the acid in the silage itself. A silage high in protein may be fed with straights or compound high in protein, which can be associated with increased lameness. Similarly a change in the amount of lameness in mid-winter may be linked to the change from one batch of silage to another.

Lameness often peaks soon after calving. It is tempting to blame this on a sudden increase in concentrate feed at calving, but Leaver (1990) concluded that there was no good evidence for this. It seems that high concentrate and low forage intake, whether achieved rapidly or slowly, is a risk factor in lameness. This will occur in early lactation, when the cow is at peak yield, and has a reduced dry matter intake, particularly if she calves down too fat. The forage part of the diet then gets reduced.

The amount of lameness in a herd may change over a period of years because of a change in feeding patterns, but alternatively extensive use of of a particular bull may be to blame. Russell (1987) found the incidence of sole ulcer, and

of white line lesions, much higher in the daughters of certain bulls when the cows were under similar management.

If lameness is encouraged by high concentrate feeding, then in those herds feeding to yield, we would expect that high-yielders would be more likely to go lame. This was suggested by Rowlands and Lucey (1986) but Merritt and others (1992) found more lameness in higher yielders in a herd on a flat-rate feeding system, which suggests that a non-nutritional factor may make high-yielding cows more prone to lameness.

A high incidence of lameness in one herd can sometimes be caused largely by a particular type of lameness, such as digital dermatitis or foul. It is important to know this, rather than simply noting a high incidence of lameness on a farm.

Effects of feeding on lameness

Starch and sugars are associated with foot lesions, and the term 'rapidly fermentable carbohydrate' (RFC) has been used. Protein, particularly rumen-degradable protein (RDP) is also linked with foot lameness. Low intake of forage or other source of fibre with high intakes of RFC is also associated with foot problems. Leaver (1990) has reviewed the earlier literature.

In the Liverpool study we measured starch and sugar content of forage and concentrates fed to over 4,000 cows in 37 farms. Analysis of the results has so far shown few consistently significant associations between feeding and lameness. Farms where a grass silage high in dry matter was fed had a highly significant reduction in the incidence and prevalence of lameness. High silage fibre and low silage protein were also linked to less lameness, but further analysis has shown that these were both linked to silage dry matter, which was the dominant factor.

Fifteen farms on flat-rate feeding were compared with 22 feeding to yield. Our hypothesis was that flat-rate would be better, since the concentrate was fed at a moderate rate over a period of time, with less fed at peak lactation. In fact the farms on flat-rate feeding had more lameness than those feeding to yield.

In 12 farms the cows were self-fed forage, or partly self-fed and partly easy-fed. In 15 the forage was all easy-fed, and in 9 forage was mixed with concentrate (complete diet). We would expect that a mixed diet would tend to reduce lameness, since a high forage intake would be encouraged, and this would encourage saliva, with its buffering capacity. In fact, there was more lameness on the farms feeding complete diets.

The reasons for the greater amount of lameness on the farms where the feeding system was the sort that we would expect to be best may be that our theories are wrong, or it could be

Fig. 1

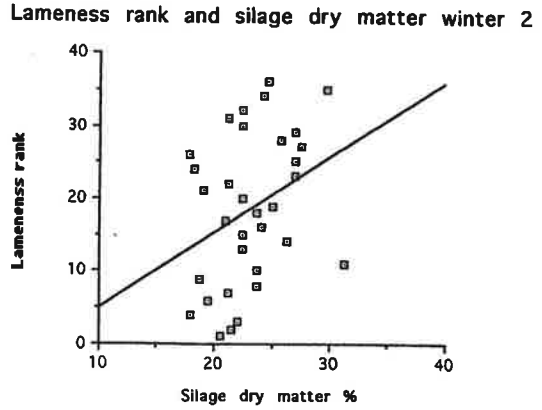
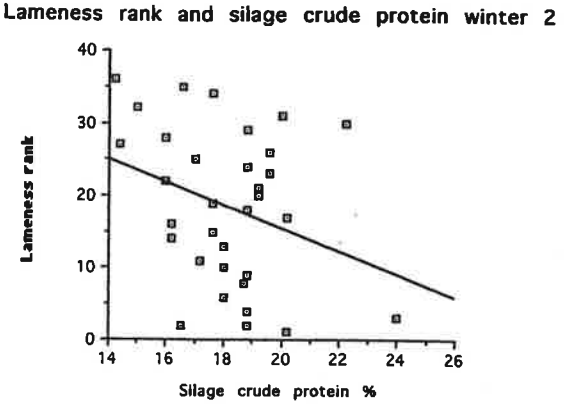


Fig. 2



that the farms opting for these feeding systems had other risk factors that were more important.

Further analysis is now in progress, to compare farms, allowing for other factors. We will also compare cows within herds. Here we will have thousands of variables, instead of 37. For example, if high-yielders in the herds feeding to yield, but not in the herds feeding flat-rate, had more lameness, then this will confirm earlier work that high concentrate feeding is a risk factor.

The reasons for the links between diet and lameness are not yet fully clear, but putting together the research findings so far, we can suggest the following:

1. Rapidly fermentable carbohydrates produce acids in the rumen. A large amount of concentrate fed in one meal, say more than 4kg, results in acid damage to the rumen wall, or to release of toxins from destruction of rumen bacteria in the low pH: toxins are found in the blood and can travel to the corium and causes lesions. The drop in pH is greater when cereals (starch) and sugars are fed, than if by-products are used, with energy from slowly fermented fibre. The pH is also likely to be more stable if the cow eats long fibre (silage, hay or straw) at the same time as the concentrate (as in complete diet) or just before or just after. Bigras-Poulin and others (1990) found that lame cows had a low milk protein in the preceding lactation. This would suggest that their energy intake had been low, which could be due to low forage intake.

2. The effect of excess RDP may be through the ammonia or possibly amines leaving the rumen, and blood urea measurement will indicate whether excess RDP is being fed.

3. The lesions commonly found in feet, particularly the outer claws of the hind feet, may be caused by all the factors mentioned, and not just feeding. Singh and others (1992a and b) found histo-pathological changes suggestive of laminitis in the feet of beef cattle, whereas in the feet of dairy cattle although there was loss of onychogenic substance there was no involvement of laminae. Both beef and dairy cattle showed haemorrhages in the sole. Greenough and Vermunt (1991) found haemorrhages (which they described as sub-clinical laminitis) in the feet of heifers before calving, as well as in older cows. Since the heifers had grown at over 0.8 kg/day on average from birth to breeding, and the faster growing heifers had the most haemorrhage, it seems reasonable to blame the lesions on the high concentrate diet.

4. It is probable that nutritional and other factors interact. For example, a heifer or cow that eats large meals of starchy concentrate and stands on concrete for 20 hours a day is likely to suffer damage to her corium.

5. The feeding system and the feed can alter the environment. For example, self-feed silage can erode non-resistant concrete so that cows are walking on sharp stones.

Feeds low in fibre or high in RFC and protein can produce liquid faeces. The cubicle house floor is then coated with slurry for the whole winter, which encourages the survival of micro-organisms, and makes the horn of the sole softer, and more easily penetrated by foreign bodies.

In conclusion, the current importance of feeding relative to other risk factors remains to be confirmed, but there are indications that housing, foot shape and stockmanship may be more important. In the future, however, cereal prices may fall. The price of by-products is likely to follow, and hence compounds and straights will be cheaper. Cows are likely to eat more concentrate, and feeding could then become a more important risk factor in lameness.

Effects of lameness on feeding.

Having looked at how feeding can affect lameness, we can now look at how lameness can affect feeding.

We commonly see that lame cows continue milking surprisingly well, but lose condition. Hassall and others (1993) showed that lame cows at grass spent less time grazing, and, surprisingly, grazed more slowly, than non-lame cows in the same herd. The effect on milk yield is unclear: Rowlands and Lucey (1986) found that heel lesions early in lactation reduced milk yields, but that sole, interdigital and wall lesions did not. Lameness later in lactation was more common in high-yielding cows - an effect of yield on lameness, perhaps, rather than of lameness on yield. Merritt and others (1992) found in one herd with a high incidence of lameness that milk yield was depressed in lame heifers, but not significantly so in older cows. Tranter and Morris (1991) found that lame cows grazing in New Zealand had lower milk yields (3,296 l) than controls (3,615 l) partly because lame cows were culled earlier (lactation length 241 vs 253 days). Butterfat and milk protein yields were also reduced, although there was little difference in the percentage of butterfat or protein.

Lameness therefore seems to reduce food intake, and hence milk yield. The effect on condition does not seem to have been precisely measured, but Tranter and Morris reported that lame cows lost 0.1 units of condition while lame, using a scale of 1-8 (not 0-5) and did not state the condition of the control cows. Lame cows do seem to lose weight, and this costs money, as they have to be fed to replace the weight. In addition, this weight loss could be one reason for reduced fertility in lame cows. Collick and others (1989) found that lame cows conceived 14 days later, than non-lame cows in the same herds. Tranter and Morris (1991) found that only 46% of their lame cows were pregnant 7 weeks after the start of mating, compared with 72% in controls.

Investigation of a lameness problem.

A veterinarian visiting a herd suffering a lameness problem with a suspected feeding component can follow this protocol.

1. The size and nature of the problem.

We have used two methods:

a) Ask everybody who lifts a cow's foot or treats a cow for lameness to record what was found, and the treatment. Lift some feet yourself to confirm the types of lesion.

b) Watch all the cows walk, and locomotion score them (Manson and Leaver, 1989). See how many cows are walking really normally (1), how many have tender feet (2) and how many are lame (3 - 5). This measure of prevalence shows the farmer the size of the problem. A single measure in the middle of summer or winter gives a valid estimate of the prevalence of lameness in that season.

2. We can then look at the epidemiology to judge the major risk factors on this farm.

Observe:

a) The system of feeding forage. If cows at a self-feed face are negotiating an electric wire, or a solid barrier too far from the clamp, or you have to pull hard to prise out the silage, forage intake will not be optimal. Easy feeding, with adequate rings or troughs, or complete diet feeding will increase forage intake. A common problem seems to be that the farmer is worried about running out of silage before turnout and restricts the silage. Ask whether some surplus silage is taken away daily for young stock or dry cows. If cows in early lactation never leave any silage, they are not being fed ad lib.

b) The system of feeding concentrates. Parlour feed dispensers can be wildly inaccurate. If more than 4kg is fed in one meal, there is a risk of rumen acidosis. If hay, straw or silage is available just before and just after the parlour feed, the rumen can be buffered.

c) The feed. Compound feeds can be based on cereals, so high in starch, or on by-products with high fibre. Phone the feed company's representative to discuss the content of a compound. A silage high in protein may be fed with a compound, or straights, also high in protein.

d) The milk. Every month our farmers receive payment based largely on the protein and butterfat in the milk. A drop in protein is a clear warning of low energy intake, which usually means low forage intake. A fall in butterfat means reduced fibre intake.

e) The cow. Condition score the dry cows. If they are above 3.0, the lower appetite after calving will reduce their total dry matter intake and hence forage intake. Note the size of the rumen on rectal palpation. This is good evidence of forage intake. The consistency of the dung can indicates fibre intake and concentrate intake. Blood samples can be helpful: high urea is an excellent indicator of intake of rumen-degradable protein relative to fermentable energy.

3. The lame cows.

As well as prompt treatment, the farmer must feed the lame cows. The loss in condition seems to be greatly reduced if the lame cows live in a straw yard with food supplied to them, rather than competing with the rest of the herd. In summer, if they can live on a nearby paddock with extra feed, or in a straw yard, this will help the condition and welfare of the cows.

Conclusion.

The practising veterinarian is in an excellent position to help advise the farmer on feeding and lameness. We do not understand all the connections, but we know enough. We do not know as much as nutritionists about nutrition, but we know more about lameness, and we can work together to advise the farmer.

Acknowledgements.

I thank Deborah Clackson, Michael Clarkson, Don Collick, Graham David, Sarah Jane Diamond, Hilary Dobson, David Downham, Barrie Edwards, Bill Faull, Sally Ann Hassall, Neil Howie, John Hughes, Felicity Manson, Jane Merritt, Julia Mill, Richard Murray, Egbert Toussaint Raven, Wanda Russell, Simrat Singh, and Jo Sutherst. Funds were provided by Ministry of Agriculture, Wellcome Trust and Universities Federation for Animal Welfare.

References

Bigras-Poulin, M, Meek AH, Martin, SW (1990) Inter-relationships among health problems and milk production from consecutive lactations in selected Ontario Holstein cows Preventive Veterinary Medicine, 8, 15 - 24

Clackson, DA, Ward, WR (1991) Farm tracks, stockman's herding and lameness in dairy cattle. Veterinary Record, 129 511 - 512.

Clarkson, MJ, Downham, DY, Faull, WB, Hughes, JW, Manson, FJ, Merritt, JB, Murray, RD, Russell, WB, Sutherst, JE, Ward, WR (1993) An epidemiological study to determine the risk factors of lameness in dairy cows. (Ref; CSA 1379). Final Report.

Collick, DW, Ward, WR, Dobson, H (1989) Associations between types of lameness and fertility. Veterinary Record, 125, 104 - 106.

David, GP (1990) Terminology and pathogenesis associated with laminitis in cattle. Proceedings VIth International Symposium on Diseases of the Ruminant Digit, Liverpool, ed RD Murray, 1-5.

Greenough, PR, Vermunt, JJ (1991) Evaluation of subclinical laminitis in a dairy herd and observations on associated nutritional and management factors. Veterinary Record, 128, 11 - 17.

Leaver, JD (1990) Effect of feed changes around calving on cattle lameness. Proceedings VIth International Symposium on Diseases of the Ruminant Digit, Liverpool, ed RD Murray, 102 - 108.

A.M. Johnston (1990) Studies of hoof horn. Proceedings VIth International Symposium on Diseases of the Ruminant Digit, Liverpool, ed RD Murray, 76 - 79.

Manson, FJ, Leaver, JD (1988) The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. Animal Production, **47**, 185 - 190.

Manson, FJ, Merritt, JB (1990) Feeding factors associated with lameness: a preliminary report from an epidemiological study. Proceedings VIth International Symposium on Diseases of the Ruminant Digit, Liverpool, ed RD Murray, 101.

Merritt, JB, Manson, FJ, Russell, WB, Downham, DY (1992) The effect of lameness on milk production in Friesian-Holstein cows. (Summary) British Society of Animal Production, March 1992, Scarborough.

Rowlands, GJ, Lucey, S (1986) Changes in milk yield in dairy cows associated with metabolic and reproductive disease and lameness. Preventive Veterinary Medicine, 4, 205 - 221.

Russell, AM (1987) The influence of sire on lameness in cows. Proceedings for 1986-87. British Cattle Veterinary Association, 213 - 219.

Singh, SS, Murray, RD, Ward, WR (1992a) Histopathology of the hooves of dairy and beef cattle in relation to overgrown sole and laminitis. (In Press) Proceedings of the VIIth International Symposium of Disorders of the Ruminant Digit, Aarlborg, Denmark, June 1992.

Singh, SS, Murray, RD, Ward, WR (1992b) Histopathological and morphometric studies on the hooves of dairy and beef cattle in relation to overgrown sole and laminitis.Journal of comparative Pathology, **107**, 319 - 328.

Singh, SS, Ward, WR, Lautenbach, K, Murray, RD (1992b) Lying behaviour of dairy cows and its relationship with lameness in

first lactation cows.(In Press) Proceedings of the VIIth International Symposium of Disorders of the Ruminant Digit, Aarlborg, Denmark, June 1992.

Singh, SS, Ward, WR, Lautenbach, K, Murray, RD (1993a) Behaviour of lame and normal dairy cows in cubicles and in a straw yard. Veterinary Record, **133**, 204 - 208.

Singh, SS, Ward, WR, Lautenbach, K, Murray, RD (1993b) Behaviour of first lactation and adult dairy cows while housed and at pasture and its relationship with sole lesions. Veterinary Record, **133**, 469 - 474.

Singh, SS, Ward, WR, Murray, RD (1992) Aetiology and pathogenesis of sole lesions causing lameness in cattle: a review. Veterinary Bulletin.**63**, 303 - 315.

Tranter, WP, Morris, RS (1991) A case study of lameness in three dairy herds. New Zealand veterinary Journal 39, 88 - 96.

Toussaint Raven, E (1989) Cattle footcare and claw trimming. Farming Press, Ipswich, UK.

THE ROLE OF TRACE MINERALS IN BOVINE LAMENESS

A. Bruce Johnson, Ph.D. & LaVerne M. Schugel, D.V.M.
Zinpro Corporation, 7825 Washington Avenue South, Suite 850, Edina, MN 55439-2441

INTRODUCTION

Bovine lameness is a problem for both the dairy and beef producer. In the beef feedlot industry, lameness is associated with reduced feed intake, reduced rate of gain and reduced feed efficiency. In the beef and dairy cow herd, lameness is associated with reduced roughage and forage intake, reduced production and reduce conception rates. This is usually the result of reduced mobility. In dairy herds, lameness is the third leading cause of culling behind reproduction and mastitis (Schearer and Van Horn, 1992). In both instances, it is usually associated with two reasons for onset. One is environmental, due to physical damage followed by infection. The other is nutritional in nature that is due to feeding practices and ration nutrient content.

DIETARY ASSOCIATIONS FOR LAMENESS

This nutritional implication and its association with bovine lameness may be the result of either ration nutrient content, feeding management practices or both.

PROTEIN CONTENT: Increasing levels of dietary protein have been indicated as possible causes of lameness. However, specific levels or types of protein have yet to be identified (Nocek, 1993).

ENERGY CONTENT: As diets progress to containing more concentrates and less roughage or if rations are fed without proper mixing and bunk management, laminitis becomes more prevalent and problematic. The incidence of laminitis increases as feedlot cattle move toward finish rations or in the first sixty to ninety days of lactation in the dairy herd.

Any time feed intake is reduced and/or concentrate or grain content of the ration increases, the potential for foot problems associated with laminitis increases.

TRACE MINERAL CONTENT: The trace mineral content of ruminant diets is also associated with preventing possible problems in bovine lameness.

IODINE: The primary physiological requirement for iodine is in the synthesis by the thyroid gland of hormones that regulate the body's rate of energy metabolism (NRC, 1988). However, iodine compounds are sometimes fed in excess of the nutritional requirement to cattle to prevent footrot (Miller and Tillapaugh, 1967).

A rate of 0.11 mg of ethylenediamine dihydriodide (EDDI)/kg BW/day was correlated with serum iodine concentrations (20-80 μg/dl) previously found to be effective in preventing footrot in cattle (Maas et al., 1989).

In another study, cattle received a capsule containing EDDI at the rate of 12.5-200 mg/animal/day (Berg et al., 1984). After 15 or 28 days, the cattle were then inoculated intradermally in the interdigital space with a mixture of *Fusobacterium necrophorum* and *Bacteroides melaninogenicus* to induce acute footrot. Control cattle had more lameness and more severe lesions than the cattle receiving the EDDI. There was a direct relationship between the dosage of EDDI and serum iodine levels. It is noteworthy to mention that the 200 mg/d level cattle showed an inability to metabolize and excrete the iodine. They had high serum iodine levels of 600-700 μg/dl of serum.

The efficacy of a salt mixture containing EDDI was studied in its relationship to preventing naturally occurring footrot in calves on pasture (Maas et al., 1984). The incidence of footrot, as measured by interdigital lesions, in the control group was 20.8%. This was significantly higher ($P < 0.05$) than the treatment group at 8.3%. The control group was fed at the rate of 0.0025% iodine, while the treatment group received 0.125% iodine in the salt mixture.

A series of studies (Preston et al., 1993) addressing iodine supplementation in feedlot diets have concluded: 1) iodine had direct antibacterial properties, but to achieve this level would be toxic to cattle; 2) serum iodine concentrations on 60-80 μg/dl are thought to be prophylactic and can be achieved by feeding levels of 60-80 mg/hd/d; 3) cellular immune response is improved by feeding EDDI and iodate; 4) blood levels reflect iodine serum levels independent of source; 5) no toxic effects were seen in feedlot cattle fed 45 mg/hd/d for 215 days or 200 mg/hd/d for 50 days.

In an early study (Berg et al., 1976), the addition of EDDI or urea to feedlot rations did not affect the frequency of experimentally induced footrot.

Severe side effects were seen after five weeks when iodine was fed at the rate of 1 mg EDDI/kg BW/d to 100 kg calves (Andersson and Tornquist, 1983).

It may be noteworthy to mention the serum protein bound iodine in cows with foot and mouth disease ranged form 1.4-4.9 µg/dl (Lal., 1981).

The recommended dietary content for iodine for lactating dairy cows is 0.6 ppm (NRC, 1988).

ZINC: The primary physiological function of zinc is as an activator and constituent in numerous enzyme systems involved in nucleic acid metabolism, protein synthesis, and carbohydrate metabolism (NRC, 1988).

The probable role of zinc in abnormal foot conditions in ruminants and the effect of zinc deficiency as a cause of foot disease in ruminants is normally associated with zinc's relationship to wound healing, and epithelial tissue repair and maintenance of cellular integrity (Weaver et al., 1978).

Groups of dairy cows on farms with high incidence of foot problems were given either 2 or 3 grams of zinc sulfate/hd for 70 days in the daily feed (Dembinski and Wieckowski, 1987). In comparison with untreated controls the prevalence and severity of the foot conditions were reduced in the treated animals. The treated animals showed increased serum carotene and serum zinc levels. In addition, the vitamin A content of the liver was increased.

We would agree with these findings and as a result, theorize that this is due to the role of zinc in enzyme activation of the conversion beta-carotene to vitamin A.

A year long study looked at the effect of zinc methionine on six categories of hoof measurements of dairy cows (Moore et al, 1989). 200 mg Zn/hd/d from zinc methionine significantly improved hoof texture and interdigital dermatitis. In addition, observations on the number of heel cracks and laminitis showed significant improvement. Ulcers and white line disease were numerically reduced as well.

Rapid and complete repair of severe interdigital lesions occurred in animals given oral zinc therapy in an effort to control infectious pododermatitis in young bull calves (Demertzis and Mills, 1973).

In a contary report, sheep fed rations medicated with zinc sulfate for up to six months did not obtain adequate control (Cross and Parker, 1981).

Zinc methionine appeared to improve hoof condition of grazing cattle in a Kansas study (Brazle, 1992). A three year study showed that the incidence of footrot was reduced by 55% from 5.38% of the control cattle affected with footrot down to 2.45%

($P < 0.06$) for the treatment cattle. The steers consumed an average of 52 mg zinc/hd/d. Cattle that did not have footrot gained 1.25 kg/d while the cattle that contracted footrot gained 1.05 kg/d. The benefit gained from the addition of zinc methionine to grazing steers appeared to be one based on nutrition.

Feedlot cattle appeared to have greater hoof strength and integrity when fed a zinc proteinate over zinc sulfate (Reiling et al., 1992). Maximum shearing force was increased by the feeding of the zinc proteinate, but interactions due to the length of time zinc proteinate was fed were noted. Time exposure to supplementation was significantly correlated to effect.

The recommended dietary content of zinc for lactating dairy cows is 40 ppm (NRC, 1988). Many nutritionists and veterinarians are recommending and feeding at least twice this level. The recommended dietary content of zinc methionine is 360 mg of zinc from zinc methionine per cow per day.

COPPER: The primary physiological role of copper is in its function as an enzyme activator and enzyme constituent (Maynard and Loosli, 1969). It also has a basic function in iron metabolism and red-blood cell maturation. A copper deficiency interferes with the synthesis of keratin leading the way for copper's role in bovine lameness. Copper needs in the immune system will also function to help prevent bovine lameness.

Lameness traced to copper deficiency and excessive intake of molybdenum and sulphate was shown by excessive enlargement of the plates of metacarpus and metatarsus in young calves (Smith et al., 1975). After 60 days treatment of daily sub-cutaneous injections of 0.5 mg/kg BW of copper glycinate, the lameness was cured and general condition of the calves improved. However, the bone enlargements remained unaffected.

Consistent lesions in the distal metacarpal and metatarsal growth plate showed radiographically as widening of the physis with irregularity and fragmentation of the metaphysis was noted in calves in a molybdenum-rich geographic area (Irwin et al., 1974.). Microscopically, focal widening of the growth plate consisted of tongues of uncalcified cartilage with delayed or impaired provisional calcification in the presence of active osteoblast. It was concluded that molybdenum induced copper deficiency in cattle is possibly not the same syndrome as primary copper deficiency. Usually primary copper deficiency is characterized by decreased osteoblast activity and osteoporosis.

The recommended dietary content of copper for lactating dairy cows is 10 ppm (NRC, 1988). Many nutritionists and veterinarians recommend feeding above this level for increased production and prevention of possible problems due to interactions with other minerals.

SELENIUM: The primary physiological role of selenium is as a constituent in glutathione peroxidase (NRC, 1988). This anti-oxidant component plays a major role in immune function resulting in its possible connection to reducing problems in bovine lameness. Signs of chronic selenium toxicosis include lameness and deformed, cracked and elongated hoofs (NRC, 1988).

It has been reported that placental retention, metritis and mastitis predispose dairy cows to foot problems. Dairy cows were fed either vitamins, selenium or both in an effort to study this interaction (Larson et al., 1980). Incidence of retained placenta, metritis, and mastitis were not statistically different among treatments. The incidence of severe foot problems by treatment group was: control-28%, vitamin A, D and E-30%, 50 mg selenium injection-48%, and both vitamins and selenium-69%. Under these research conditions, selenium and vitamin treatments were not effective in reducing the incidence of retained placentas and may have potentiated ulcerative foot disease.

Currently the maximum allowable dietary level of selenium for lactating dairy cows is 0.3 ppm (NRC, 1988).

MANGANESE: The primary physiological role of manganese is an activator of a number of enzyme systems. In addition, it plays a role in reproduction (NRC, 1988). A deficiency of manganese results in suppression of estrus and reduced conception rate. Poor skeletal growth and weak, poor condition of legs, feet and joints are noticed (Mertz, 1987). Manganese deficient animals were found to exhibit skeletal abnormalities in a number of animal species; however, manganese has not been considered as necessary for wound healing (Klimis-Tavantzis, 1994).

Investigations disclosed that the role of manganese was necessary for the activities of several enzyme systems involved in the synthesis of the bone matrix. As in the process of bone formation, wound healing contains high levels of glycosaminoglycans. A manganese deficiency would retard the synthesis of these glycosaminoglycans, and therefore, retard wound healing. Any problems with wound healing or bone formation might possibly manifest itself in the

problems of bovine lameness.

The recommended dietary content of manganese for lactating dairy cows is 40 ppm (NRC, 1988).

TOPICAL APPLICATIONS OF TRACE MINERALS

Many producers rely on the topical application of zinc and copper in the treatment and control of footrot.

Zinc sulphate footbaths at a concentration of 20% proved useful in controlling the spread of interdigital dermatitis and as therapy of mild forms of the condition (Nutter and Moffitt, 1990).

The use of a 5% copper sulfate footbath for one week, then a quaternary ammonium detergent helped control the outbreak of seborrhoeic dermatitis (greasy heel) in dairy cattle (McLennan and Daniel, 1991).

Topical application of copper sulfate required 33 days and 6-8 dressing changes before healing an experimentally induced wound on the claw of dairy cattle (Szymonis-Szymanowski, 1987).

INTERACTION WITH BACTERINS

An aluminum hydroxide adjuvanted bacterin (Miles Inc., 1986) containing two strains of *Fusobacterium necrophorum* for use as an aid in the prevention and treatment of footrot in sheep and prevention of acute footrot in cattle carries a mineral feeding statement. It states "For best results, sheep should be receiving free choice mineral providing 40 to 80 mg of zinc as zinc methionine per head per day."

HOOF TRACE MINERAL ANALYSIS

Results of hoof trace mineral analysis show a strong correlation between zinc and copper. Copper concentrations significantly decreased during pasturing season. This change may be related to dietary change or management factors associated with grazing and forage availability (Hidiroglou and Williams, 1986).

SUMMARY

To better understand the role of trace minerals in bovine lameness, nutritionists and veterinarians need to understand the function and interrelationship of each of these trace minerals. In addition, they need to know how each mineral relates to foot problems and the immune process. They must know if changing one level of one mineral will create a problem associated with another mineral and design feeding programs to prevent these potentially dangerous circumstances. Ultimately, they must understand the component systems of mineral level and different mineral sources.

REFERENCES:

Andersson, L. and M. Tornquist. 1983. Veterinary Record 113(10):215-216.

Berg, J.N., L.N. Brown, P.G. Ennis and H.L. Self. 1976. Am. J. Vet. Research 37(5):509-112.

Berg, J.N., J.P. Maas, J.A. Paterson, G.F. Krause and L.E. Davis. 1984. Am. J. Vet. Research 45(6):1073-1078.

Brazle, F. 1992. Feedstuffs, Vol. 64, No. 42.

Cross, R.F. and C.F. Parker. 1981. J. Am. Vet. Med. Assoc. 178:704.

Dembinski, Z. and W. Wieckowski. 1987/1988. Bulletin of Vet. Institute in Pulawy 30-31 (1-4):104-112.

Demertzis, P.N. and C.F. Mills. 1973. Veterinary Record (No.8):219-222.

Hidiroglou, M. and C.J. Williams. 1986. Am. J. Vet. Research 47(2) P. 301-3.

Irwin, M.R., P.W. Poulos, Jr., B.P. Smith, and G.L. Fisher. 1974. J. Comparative Pathology 84(4):611-621.

Klimis-Tavantzis, D.J. 1994. CRC Press.

Lal, L.G.S. 1981. Indian Vet. Journal 58(8):646-648.

Larson, L.L., F.G. Owen, P.H. Cole and E.D. Erickson. 1980. J. An. Sci. 51(Suppl.1):296.

Maas, J. L.E. Davis, C. Hempstead, J.N. Berg and K.A. Hoffman. 1984. Am. J. Vet. Research 45(11) P.2347-50.

Maas, J., J.N. Berg and R.G. Petersen. 1989. Am. J. Vet. Research 50(10):1758-1759.

Maynard, L.A. and J.K. Loosli. 1969. Animal Nutrition 6th Edition.

McLennan. M.W. and R.C.W. Daniel. 1991. Austrilian Veterinary Journal 68(2):76-77.

Mertz, W.M. 1987. Academic Press, Inc.

Miles, Inc. 1986. Animal Health Products, Shawnee Mission, Kansas.

Miller, J.K. and K. Tillapaugh. 1967. Cornell Feed Serv. 62:11.

Moore, C.L., P.M. Walker, J.R. Winter, M.A. Jones and J.W. Webb. 1989. Transactions of Illinois State Academy of Science P. 99-108.

National Research Council (NRC). 1988.

Nocek, J.E. 1993. W.D. Hoards & Sons Company.

Nutter, W.T. and J.A. Moffitt. 1990. Veterinary Record 126(8):200-201.

Preston, R.L., S.J. Bartle and M.M. Siddiqui. 1993. Texas Tech Research Results, Lubbock.

Reiling, B.A., L.L. Berger, G.L. Riskowski and R.E. Rompala. 1992. J. An. Sci. 70(Suppl.1):313.

Shearer, J.K. and H.H. Van Horn. 1992. University of Florida.

Smith, B.P., G.L. Fisher, P.W. Poulos, M.R. Irwin. 1975. J. Am. Vet. Med. Assoc. 166(No.7):682-688.

Szymonis-Szymanowksi, W. 1987. Polskie Archiwum Weterynaryjne 27 (2-3):61-73.

Weaver, A.D., Raven, E. Toussaint, J.R. Egerton, P.R. Greenough, P.N. Demertzis, D.J. Peterse and A. Modrakowski. 1978. Skara, Sweden; Veterinary Institute P. 113.

MINERAL AND SELECTED VITAMIN NUTRITION RELATED TO LAMENESS

Marion E. Smart, D.V.M., Ph.D., Department of Veterinary Internal Medicine, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan S7N OWO

INTRODUCTION

The impact that minerals and vitamins have on skeletal and muscular integrity is complex. The veterinarian's traditional method of viewing nutritional problems in isolation does not take into consideration the complex interactions among genetics, the environment, metabolism, nutrition and nutrient balance. I believe that an "academic" approach is important for us to understand, diagnose, and correct the impact that these interactions have on skeletal and muscular homeostasis. As livestock breeding, feeding, and management becomes more sophisticated through advances in biotechnology and constrained by environmental and animal welfare issues, the ability of the veterinarian/nutritionist to identify and correct often subtle nutritional imbalances is important.

BIOAVAILABILITY/INTERACTIONS

Bioavailability is often considered synonymous with digestibility. In a broader sense, bioavailability encompasses nutrient solubility, absorption, digestibility, organ uptake and release, enzymatic transformation, secretion and excretion. These metabolic events are further influenced by genetics, age, nutrient interactions, metabolic state, nutritional reserves, and hormonal controls (Brooner, 1993).

Interactions contribute to nutrient bioavailability. These interactions can occur at several sites within the feeds, the lumen of the gastrointestinal tract, at the site of absorption or transportation, within cell metabolism (enzymes function), and through accelerated or suppressed excretion (Davis, 1980).

NUTRIENTS AND SKELETAL DEVELOPMENT

The growth pattern and remodelling of bone can be altered by any factor that impairs:

1) Cell differentiation and multiplication

2) The formation of the organic matrix (collagen, chondroitin sulfate-rich proteoglycans)

3) The mineralization of this organic matrix

Nutrition plays an important role at all three levels. The role of nutrition in skeletal growth has been extensively studied. Recent studies that involve growth and inducing or differentiation factors give us detailed knowledge of what is occurring at the cellular level (Seyedin and Rosen, 1992). An excessive or deficient nutrient intake is often complicated by nutrient interactions or minor deficiencies of other nutrients. These factors also contribute to the different clinical signs, that can seen in primary vs. secondary deficiencies.

Bone can only develop/remodel normally if the necessary ingredients are present and the required synthetic pathways operative

(Rodan 1992). Mechanical load bearing has a direct requirement for a particular amount of bone tissue with material properties that can withstand a functional load. Nutrition can alter the ability of the bone to withstand the mechanical load. The end result can be skeletal deformities, fractures or growth retardation.

Osteopenia is defined as decreased bone density, this can be osteoporosis, a loss of bone matrix and mineral or osteomalacia a decrease in bone mineral but not matrix.

Bone plays not only an essential role in support and movement but also in the regulation plasma calcium homeostasis (Baine and Watkins, 1993). In the adult, the impact of an altered nutrient intake on the skeleton is small over the short term. In the calf, nutrition has a significant role during the active growth phase of the skeleton, as bone turnover rate is very high. In the young adult, 3 to 5% of the total skeletal tissue is remodelling at one time. Although dietary restriction of certain nutrients may cause a drain on the skeletal reserve, growth may be retarded or other clinical signs seen before overt skeletal abnormalities occur.

NUTRITION AND THE GROWTH PLATE (Hill, Ruth, Van Sickle et al, 1987)

In the young animal, the growth plate is important, especially when lameness associated with metabolic bone disease occurs. The primary growth plate is responsible for longitudinal bone growth and the secondary growth plate (ossification centre) is responsible for endochondral ossification of the epiphysis (Poole 1992).

The primary growth plate consists of a cartilaginous zone, where chondrocytes mature in an orderly state. The matrix is prepared for calcification in these zones and chondrocyte maturation proceeds in this permissive environment.

Anywhere along the growth plate, a disrupted internal cellular or external environment can abort or disrupt normal maturation. If growth plate maturation is altered at any level, widening and irregularities of the growth plate can occur, islands of poorly mineralized growth plate may be evident in the metaphysis, and micro fractures at the level of the primary trabeculae can occur.

Clinically, the calf may be moderately to severely lame, have enlargement of the growth plate particularly evident in the distal metacarpus, tarsus and costochondral junction. Valgus or varus limb deformities may develop. Spontaneous fractures of the long bones, ribs, and vertebral bodies can result in the calf being down or showing neurological signs. If the articular cartilage is involved lameness and synovial joint distension can occur.

CARTILAGE AND BONE METABOLISM

Collagen

Collagen makes up one third of the total protein in the body, 57% of which is in the bone. Collagen is rich in glycine, proline and lysine. Collagen maturation occurs rapidly, once the collagen crosslinkages are stabilized mineralization proceeds. Nutritional interference with collagen

synthesis and maturation can occur at various levels:

1) Decreased uptake and hydroxylation of proline occurs with vitamin D, vitamin C, and zinc deficiency.

2) Decreased crosslinkages of the polypeptide chains occurs with vitamin E, copper and zinc deficiency.

Mucopolysaccharide

Chondroitin sulfate-rich proteoglycan is the extracelluar interfibrous component, which acts as a cement substance in which collagen fibres are embedded. Manganese is essential for the activity of glycosyltransferase, an enzyme involved in chondrotin sulphate synthesis. Decreased uptake of sulphate has been reported in zinc deficient rats. Alkaline phosphatase, a zinc metalloenzyme may be involved in matrix formation. Inadequate matrix calcification has been attributed to a decrease in sulphate groups.

Calcification

Calcification begins once a mature organic intercellular matrix is formed. Three zones can be distinguished: the crystal lattice interior, the crystal surface and the hydration shell. All zones are involved in ion exchange. The interior has the slowest rate of ion turnover and is the site of sequestration of strontium, radium, and lead. Fluoride forms a very stable crystal.

SPECIFIC MACROMINERALS AND THEIR ROLE

Calcium (Ca)

Seventy percent of the plasma calcium relies on the physicochemical properties of bone and a minimal level of bone cell activity. Above or below, a base concentration, plasma calcium is controlled by parathyroid hormone (PTH), calcitonin, and vitamin D metabolites.

Lysine, arginine, lactose and vitamin D can increase calcium absorption. Oxalate, phytate, fibre, and saturated fats, if not altered in a functional rumen, can decrease calcium absorption . Substances that increase abomasal pH can decrease Ca absorption. Calcium status alters the efficiency of calcium absorption. The efficiency of absorption declines with age, with the deficit made up by increased bone resorption (Scott, 1981).

Diets high in anions (acidic) are used to prevent milk fever. These diets produce an increased pool of exchangeable Ca, by bone resorption. If these diets are misused and cause a subclinical metabolic acidosis, osteoporosis may develop (Romo et al, 1991).

Calcium deficiency, in the adult, stimulates the generation of the resorptive remodelling units, through parathyroid hormone. These units are primarily within the cortex and on endosteal surfaces and cause cortical thinning and porosis. In the calf, there is failure of adequate mineralization of the zone of hypertrophying cartilage, and inadequate mineralization of the bone matrix. A high dietary intake of phosphorous associated with a low calcium intake can

cause secondary hyperparathyroidism (Calvo, 1993).

A chronic excess of dietary calcium in bulls leads to hypercalcitonism (C-cell hyperplasia or neoplasia) arrested bone resorption and osteopetrosis in an attempt to regulate serum calcium (Krook, Lutwak, McEntee et al, 1971).

Phosphorus

Phosphorus (P) is the second most abundant mineral, 85% of which is found in bone. The salivary gland not the kidney is the major route of P excretion, in the ruminant (Yano, Yano, and Breves, 1991). Thus saliva plays a major in phosphorous homeostasis. The efficiency of phosphorus absorption can be altered by age, source of phosphorous, Ca:P ratio, vitamin D status, rumen and intestinal pH, other minerals, and dietary fat (Morse et al, 1992).

In cattle, phosphorus depletion occurs primarily with an inadequate dietary intake. Highly leached acid soils and alkaline soils are associated with phosphorus deficiency (Schryver and Hintz, 1978). A dietary intake of 5.1 to 6.6 g of P/day to Hereford heifers, within 6 months produced clinical signs of weight loss, reduced feed intake, reluctance to move, abnormal stance and spontaneous bone fractures. When they were fed a diet containing 11.7 to 20.5 g P /day, their health was restored (Call et al, 1986).

In a complex trial, over 10 years, female Herefords were monitored from weaning to aged cows. Shupe, Butcher, Call et al, 1988 found that a dietary intake of < 6 g P/day results in improper mineralization of

osteoid tissue with severe clinical signs of poor growth and spontaneous fractures. Phosphorous supplementation > 12 g/day resulted in a rapid clinical improvement. An osteodystrophy related to a phosphorous deficiency is described in yearling Angus steers grazing a winter pasture of swede (Brassica napus), in New Zealand (Thompson,1987).

A secondary phosphorus deficiency can be caused by the ingestion of an excess of iron, aluminium, beryllium, calcium, magnesium, strontium and molybdenum (Schryver and Hintz, 1978). A dietary intake of 0.02% aluminium can severely affect P metabolism and performance in growing calves, by reducing P absorption. This affect is more severe in low phosphorus diets (Crowe et al, 1990). Aluminum is in the soil and can affect grazing animals. Dietary P availability is higher in dairy calves fed a strong anionic diet (-14.2 meq/100 g diet DM) (Beighle et al. 1988).

VITAMINS AND BONE METABOLISM

Vitamin D

The calciferols (Vitamin D and its metabolites) are fat-soluble biologically active secosteroids (Allen and Weingand 1985). Vitamin D function is closely connected to that of Ca, P, calcitonin, and PTH (Allen and Weingand, 1985). A decrease in extracelluar calcium concentration stimulates PTH secretion which stimulates renal production of 1,25 (OH)2 Vit D3 which enhances the intestinal absorption of Ca by production of a D3 dependent calcium binding protein. Intestinal phosphate transport is

also enhanced by an unknown mechanism (Allen and Weingand, 1985).

Vitamin D recruits bone marrow stem cells for new bone formation and remodelling (Suda, Shinki and Takahashi, 1990). 24,25(OH)2 D3 and 1,25 (OH)2 D3 has a stimulator effect on proteoglycan and type II collagen synthesis (Poole, 1992). There is a seasonal and geographical variation in UV activation of provit D in the skin (Henry and Norman, 1984).

Vitamin A

Vitamin A is involved with the synthesis of specific glycoproteins that control cellular differentiation. Although not a direct cause of lameness, vitamin A does, play a role in bone metabolism. If vitamin A is deficient there is diminished uptake of sulphur in the formation of chondroitin sulphate, cartilage cells do not follow the normal pattern of growth, maturation, and degeneration. Zinc deficiency can result in a failure of the release of vitamin A from the liver, through the role of zinc in retinol binding protein. This can result in an apparent vitamin A deficiency.

Vitamin A in excess may impair the ability of periosteal progenitor cells to differentiate into osteoclast, thus subperiosteal reabsorption of bone fails. Thus there is reduced bone reabsorption and increased osteoblastic activity.

Vitamin K (Price, 1988)

Bone Gla protein (osteocalcin), a vitamin K dependent protein, is secreted by osteoblast and binds to mineral. This protein

also plays a critical role in bone reabsorption and is regulated by vitamin D3. Serum osteocalcin concentrations are used as an indicator of bone mobilization.

Biotin

Biotin has been reported to improve the integrity of the hoof horn in cattle. The metabolic role of biotin is in carbohydrate and lipid metabolism (Dakshinamurti, 1988). Biotin is unstable in oxidizing conditions, thus processing of feed stuffs can result in substantial losses of biotin. The bioavailability of biotin in most feeds is low. Most biotin available to the animal is through gut microbial synthesis.

NUTRIENT DEFICIENCIES RELATED TO MUSCLE INTEGRITY

Nutritional Myopathy

Nutritional myopathies occur as a result of selenium and Vitamin E deficiency. The clinical signs and response to therapy depends on the age of the animal, the muscle groups affected, and the identification of dietary factors (Table 1) and environmental stressors (Table 2).

Pathogenesis

Both selenium and Vitamin E protects the cell from free radical injury. Selenium functions at the cytosol level through Glutathione peroxidase (GSH-Px). Lipid soluble vitamin E prevents lipid peroxidation of the polyunsaturated fatty acids in cell membranes. If one or both these systems are compromised in striated

muscle an intracellular cascade of events occurs related to the damage caused by unchecked free radical production.

Clinical Signs

Perinatal

Still born or weak calves can occur. If the skeletal muscles are involved the calf will be stiff and sore and the affected muscles will be painful and firm. Dyspnea and abdominal breathing is seen if the intercostal muscles and the diaphragm are involved. If the tongue is involved the calf will be unable to suck. Sudden death can occur if the myocardium is involved.

Young Adults

An acute azoturia syndrome is seen. The muscles primarily involved are the back and thigh muscles (Chalmers et al, 1979). Subclinical myopathy was diagnosed in the group of feedlot cattle based on elevated serum CPK and AST concentrations and a myopathy seen in biopsy specimens (Smith, Palmer, Hulland et al, 1985).

Mature

A milk fever like syndrome has been described, these cows did not respond to conventional milk fever therapy. The authors attribute the problem to the stress of repeated dietary changes, a digestive upset and low Se and vitamin E intake (Gitter and Bradley, 1978). In one case report pregnant Chianina heifers experienced abortion and periparturient recumbency. This was associated with a combined deficiency, rapid growth and the

stress of pregnancy and calving (Hutchinson, Scholtz and Drake, 1982).

Clinical Pathology

The plasma activity of several enzymes of muscle origin are used as a diagnostic tool and to monitor the response to treatment (Anderson, Berrett and Patterson, 1976). The concentrations in the plasma vary dependant on the severity and location of the muscle damage and the half life of the enzyme.

Plasma activity of creatine phosphokinase (CPK) is significantly correlated with microscopic damage of the muscle and is a useful tool in monitoring recovery (Smith, 1994) and identifying subclinical animals (Allen, Bradley, Berrett et al 1975).

Plasma Se concentrations are used as a measurement of Se status. Erythrocyte GSH-Px is also a stable measure of Se status, but depends on the availability of Se during erythropoiesis (Koller and Exon, 1986). Calf plasma Se concentrations at birth are significantly correlated with that of the dams at parturition (Hidiroglou, Batra and Roy, 1994).

Plasma or serum vitamin E concentrations are useful indicators of status, as the body has limited stores. Liver is the principle site of vitamin E storage. These stores vary dependent on the season of the year, physiological state and breed of the animal and the dietary intake (Charmely et al.1992). Plasma concentrations are low in calves independent of the dams' status (Hidiroglou, Batra and Roy, 1994).

CONCLUSION

The veterinarian/nutritionist when investigating a case of nutritionally induced lameness must take into account the complex nature of the problem. An accurate evaluation of the past and current feeding practices must be done. Properly taken feed and water samples should be analyzed and these values should be used in the final ration evaluation.

Feed bunk management is important to access feed intake and wastage, ingredient sorting and adequate space. Housing, climatic and environmental conditions, and animal comfort and body condition are all factors that can impact on nutrient intake. Metabolic profiles can be done, but may be of limited value as they reflect only one point in time and some of the parameters are under tight homeostatic control.

Once all the data is collected and analyzed, new rations can be formulated using published nutrient requirements for the class of animals involved (Table 3). One must keep in mind that these values are only guides and must be adjusted based on experience, the breed, age and body condition of the animals affected, dry matter intake, diet energy density, environmental conditions, and dietary interactions present. Recommendations should take into consideration, your client's ability to follow through with your advise, with the feeds on hand.

The animals' response should be closely monitored after the diet has been corrected. If no response is observed within a reasonable period of time, then your client's compliance and the entire system must be

reevaluated. Something may have been over looked in the initial examination. In some nutritional problems a limited response maybe related to permanent metabolic or structural changes, and in some cases the response may be delayed. Your client should be made aware of these problems.

REFERENCES

Agricultural Research Council. 1980. The nutrient requirements of ruminant livestock. Commonwealth Agricultural Bureaux, Gresham Press, Surrey.

Allen W.M., Bradley R., Berrett S. et al. 1975. Degenerative myopathy with myoglobinuria in yearling cattle. Br. vet. J 131:292-308.

Allen T.A. and Weingand K. 1985. The vitamin D (calciferol) endocrine system. Comp Con Ed 7:482-488.

Anderson P.H., Berrett S. ,and Patterson D.S.P. 1976. The significance of elevated plasma creatine phosphokinase in muscle damage of cattle. J Comp Path. 86:531-538.

Baine S.D. and Watkins B.A. 1993. J Nutr 123:317-322.

Beighle D.E., Tucker W.B.,and Hemken R.W. 1988.Interactions of dietary cation-anion balance and phosphorous: Effects on growth and serum inorganic phosphorous in dairy calves. J Dairy Sci 71:3362-

Bronner F. 1993. Nutrient bioavailability, with special reference to calcium. J Nutr 123:789-802.

Call J. W., Butcher J. E., Shupe J. L. et al.1986. Dietary phosphorus for beef cows. Am J Vet Res 47:475-481

Calvo M. S. 1993. Dietary phosphorus, calcium - Metabolism and bone. J Nutr 123:1627-1633.

Chalmers G.A., Decaire M., Zacher C.J. and Barrett M.W. 1979. Myopathy and myoglobinuria in feedlot cattle. Can vet J 20:105-108.

Charmley E., Hidiroglou N., Ochoa L. et al. 1992. Plasma and hepatic alpha-tocopherol in cattle following oral or intramuscular supplementation. J Dairy Sci 75:804-810.

Crowe N. A., Neathery M. W., Miller W. J. et al. 1990. Influence of high dietary aluminum on performance and phosphorus bioavailability in dairy calves. J Dairy Sci 73:808-818.

Dakshinamurti K. and Chauhan J. 1988. Regulations of biotin enzymes. Ann Rev Nutr 8:211-233.

Davis G.K. 1980. Microelement interactions of zinc, copper, and iron in mammalian species. Ann New York Acad Sci :130-139.

Gitter M., Bradley R., and Pepper R. 1978. Nutritional myodegeneration in dairy cows. Vet Rec 103:24-26.

Hill M.A., Ruth G.R., Van Sickle D.C. et al. 1987. Histochemical morphologic features of growth cartilages in long bones of pigs of various ages. Am J Vet Res 48:1477-1484.

Koller L.D. and Exon J.H. 1986. The two faces of selenium - deficiency and toxicity - are similar in animals and man. Can J vet Res 50 :297-306.

Henry H.L. and Norman A.W. 1984. Vitamin D: Metabolism and biological actions. Ann Rev Nutr 4:493-520.

Hutchinson L.J., Scholz R.W., and Drake T.R. 1982. Nutritional myodegeneration in a group of Chianina heifers. J Am vet Med Ass 181:581-584.

Krook L., Lutwak L., McEntee K. et al. 1971. Nutritional hypercalcitoninism in bulls. Cornell Vet 61: 625-639.

Morse D., Head H.H., Wilcox C.J. et al.1992. Effects of concentration of dietary phosphorous on amount and route of excretion. J Dairy Sci 75:3039-3048.

Nutrient requirements of beef cattle. 1984. National Academy Press, Washington D.C.

Poole A.R. 1992. The growth plate: Cellular physiology, cartilage assembly and mineralization. Cartilage:Molecular Aspects Ed. B.K.Hall and S.A. Newman. CRC Press, Inc. 179-211.

Rodan G.A. 1992. Introduction to bone biology. Bone 13:S3-S6.

Romo G.A., Kellems R.O., Powell K. et al. 1991. Some blood minerals and hormones in cows fed variable mineral levels and ionic balance. J Dairy Sci 74:3068-3077.

Rice P.A. 1988. Role of vitamin K dependent proteins in bone metabolism. Ann Rev Nutr 8:565-583.

Schryver H.F. and Hintz H.F. 1978. Handbook series in Nutrition and Food. M. Richeigh Ed.Section E Nutritional disorders Vol II Effect of nutrient deficiencies in animals CRC Press.

Scott D. and McLean A.F. 1981. Control of mineral absorption in ruminants.Proc Nutr Soc 40:257-266.

Seyedin S.M. and Rosen D.M. 1992. Cartilage growth and differentiation factors. Cartilage: Molecular Aspects. Ed. B.K. Hall and S.A. Newman. CRC Press Inc.:131-151.

Shupe J.L., Butcher J.E., Call J.W. et al. 1988. Clinical signs and bone changes associated with phosphorous deficiency in beef cattle. Am J Vet Res 49:1629-1636.

Shupe J.L., Christofferson P.V., Olson A.E. et al.1987. Am J Vet Res 48.1498-1503.

Smith D.L., Palmer S.T., Hulland T.J. et al. 1985. A nutritional myopathy enzootic in a group of yearling beef cattle. Can Vet J 26:385-390.

Smith G.M., Fry J.M., Allen J.G. and Costa N.D. 1994. plasma indicators of muscle damage in a model of nutritional myopathy in weaner sheep. Aust Vet J 71:12-17.

Suda T., Shinki T., and Takahashi N. 1990. The role of vitamin D in bone and intestinal cell differentiation. Annu Rev Nutr 10:195-211.

Thompson K.G. and Cook T.G. 1987. Rickets in yearling steers wintered on a swede (Brassica napus) crop. NZ vet J 35:11-13.

Yano F., Yano H.,and Breves G. 1991. Calcium and phosphorus metabolism in ruminants. Physiological Aspects of Digestion and Metabolism in Ruminants: Proceedings of the seventh Inter Symp Rum Physio. Acad Press Inc:277-291.

TABLE I. Dietary stressors that impact on Vitamin E and Selenium Availability.

General:	Dietary upsets	
Increase Oxidative Load:	1)	Cupric or ferric ion
	2)	High dietary PUFA
	3)	Lipids in lush pastures
	4)	Rancid fats
	5)	Mycotoxins (Zearalone)
	6)	High Vitamin A
Decrease Se:	1)	Antagonist - 3 gms Zn; 100 mg Ca; 500 mg Cu; 5 gm Fe; 5 gm S/kg DM
	2)	Inorganic Se → insoluble form by rumen microorganisms
	3)	Forage storage, silage vs. hay
	4)	Soil/Plant/Environmental Interactions - grey wooded soils - heavy rainfall - acidic soils/iron oxide - high sulphur
	5)	Replace plant protein source with non-protein nitrogen
Decrease Vitamin E:	1)	Storage/high moisture/acid environment
	2)	Propionic acid
	3)	Weathered forages
	4)	Feed processing
	5)	Nitrates
	6)	High C18:3 fatty acid
	7)	Injectable unesterified α-tocopheral better than acetate ester

Table II. Environmental Stressors that can Potentiate a Vitamin/Selenium Deficiency in Cattle.

Sudden unaccustomed exercise

Sudden weather changes

Transportation Stress

Table III. Ca, P, Vitamin A & D Requirements for Cattle Under One Year of Age Based on NRC and ARC Figures.

Class		Weight Kg	Gain Kg	DM I Kg	Ca %	Ca gm/Kg BW	P %	P gm/Kg BW	Se. mg/kg DM	Vit. A. IU/kg DM	Vit. D. IU/kg DM	Vit. E. IU/kg DM
Mid-frame Steers	NRC	136	1.4	4	1.13	0.3	0.47	0.2	0.2	455	275	15-60
	ARC					0.25		0.15	0.1			15-23
Med. Frame Steers	NRC	318	1.4	7	0.49	0.09	0.26	0.06				
	ARC					0.10		0.66				
Large Frame Steers		136	1.4	4.4	0.99	0.32	0.39	0.13				
Large Frame Steers		318	1.4	8.2	0.45	0.12	0.26	0.7				

* The same requirements for all classes.

NUTRITION, RUMEN ENVIRONMENT AND LAMINITIS

Randy D. Shaver, Ph.D.
Department of Dairy Science
College of Agricultural and Life Sciences
University of Wisconsin
Madison, WI 53706

Introduction

Laminitis in dairy cattle causes economic loss related to treatment costs and higher cull rates. However, the total economic impact of laminitis is greater because of adverse effects on intake, milk yield, and reproduction. Excessive grain or nonstructural carbohydrate (NSC) feeding, slug feeding of grain (feeding grain only 2X daily), feeding sources of NSC that are rapidly fermented in the rumen, and feeding finely chopped silages are causative factors in the development of laminitis because of their propensity for causing ruminal acidosis (Nocek, 1985).

Livesey and Fleming (1984) reported an increase in clinical laminitis from 2 of 26 to 17 of 25 as concentrate to forage ratio (C:F) was increased from 50:50 to 60:40. Wheat and barley, which are rapidly fermented in the rumen, were the sources of NSC in this trial. Manson and Leaver (1988) observed an increase in lameness over weeks 3 to 22 of lactation as C:F was increased from 44:56 to 58:42 with barley as the source of NSC. A similar trend was observed by Manson and Leaver (1989) as C:F was increased from 40:60 to 60:40 with barley as the source of NSC.

The purpose of this paper is to review the role of nutrition and feeding management in the development of ruminal acidosis, and discuss feeding guidelines for the prevention of laminitis.

Prepartum Adaptation

Bazeley and Pinsent (1984) evaluated the effects of social, environmental, and nutritional changes at calving on the incidence of laminitis in dairy cattle. Herds with a high incidence of laminitis tended to put cows through more abrupt changes at calving than low incidence herds. This underscores the need for specific feeding and management programs that allow prepartum and early postpartum cows to adapt gradually to social, environmental, and nutritional changes.

National Research Council (NRC;1989) estimates dry matter (DMI) of dry cows at 1.8 to 2.0% of BW, or 12.5 to 13.5 kg for a 680 kg dry cow. However, intakes are not maintained at this level for the full dry period, as several trials reported a gradual decline (5% per week) in DMI starting 2 to 3 weeks prior to calving followed by a rapid drop (30%) starting 3 to 5 days prior to calving (Shaver, 1993). Van Saun (1992) reported a 15% decline in DMI between the close-up (last 2-4 weeks) and far-off (first 4-6 weeks) dry periods. DMI decline of 5% between weeks 3 and 2 prepartum, 5% between weeks 2 and 1 prepartum, and 30% during the last week prepartum approximates a 15% average reduction between the close-up and far-off dry periods.

This prepartum intake decline dictates that a separate ration with higher nutrient densities be fed to close-up dry cows to more closely meet their nutritional requirements. Assuming a far-off dry group DMI of 1.8% of body weight and a 15% reduction in DMI between the far-off and close-up dry groups, DMI for the close-up dry group would average 10.0 to 10.5 kg per cow per day. This level of intake can be used to formulate nutrient densities of close-up dry group rations.

Lead feeding concentrates to close-up dry cows is an important practice to improve energy intake in cows confronted with prepartum depression of intake, and to adapt cows gradually to the higher starch content of lactation rations. Dirksen et al. (1985) reported that lead feeding concentrates prepartum to increase starch intake and thereby increase ruminal volatile fatty acid (VFA) concentration increases the absorptive capacity of the ruminal papillae, and suggested that this was important for reducing ruminal acidosis and intake lag in the early postpartum cow. Also, prepartum lead feeding of concentrates provides an adaptation period for the rumen microbial population as it shifts from a high fiber dry cow ration to a higher starch lactation ration.

Knowledge of prepartum intake decline should cause us to re-evaluate some of our lead feeding guidelines. Some field recommendations call for gradually adding concentrates to dry cow rations starting 2 to 3 weeks prior to calving, at levels up to 1% of BW as concentrate DM. This could result in feeding 80% concentrate and 20% forage during the last week prepartum. Limiting concentrate DMI to less than .75% of BW in the close-up dry period should provide a reasonable adaptation program, considering that prepartum intake decline limits total DMI of the close-up dry group to about 1.5% of BW. This would limit concentrate intake to about 50% of ration DM, or 5 kg per cow per day for a close-up dry group with a total DMI of 10.0 to 10.5 kg per cow per day. It should be noted that concentrate refers to all non-forage ingredients in the ration. One major advantage of feeding a total mixed ration (TMR) to close-up dry cows is that it provides the opportunity to maintain a constant C:F ratio as DMI fluctuates dramatically during the week prior to calving.

Close attention should be given to the environment in which close-up dry cows are housed and fed. Cow comfort, ventilation, and stall, lot and feed bunk management are particularly important for close-up dry cows under stress associated with prepartum intake decline and calving. Whenever practical, use the close-up dry period to adapt cows to their early postpartum environment. It appears that cows which undergo abrupt environmental and social changes around the time of calving are more prone to metabolic disorders (Bazeley and Pinsent, 1984; Pehrson and Shaver, 1992). This requires especially careful management of first lactation heifers that are being introduced to new herdmates and the milking barn for the first time. Prepartum management is just as important as prepartum nutrition!

Feeding Lactating Cows

Dry Matter Intake

Peak milk yield occurs before peak DMI in early lactation. Kertz et al. (1991) found that intake data were best fit when separate equations were developed for each week postpartum up through week five. Separate equations were also developed for the following periods: weeks 6-8, 9-13, and 14-20 postpartum. DMI did not peak until about 10 weeks postpartum for both multi- and primiparous cows.

DMI was calculated for weeks 1-8 postpartum assuming 636 kg week 1 BW, 51 kg BW loss over the 8 week period and 41 kg 4% FCM for multiparous cows, and 545 kg week 1 BW, 29 kg BW loss over the 8 week period and 30 kg 4% FCM for primiparous cows. Estimated intakes are summarized below:

	Multiparous Cows	Primiparous Cows
	kg DMI/day	
•Week 1	16.6	14.1
•Week 2	19.3	15.9
•Week 3	21.2	17.3
•Week 4	22.3	18.2
•Week 5	23.9	18.9
•Week6-8	24.8	19.7

DMI of an early postpartum group at these production levels and averaging 4 weeks postpartum with 65% multi- and 35% primiparous cows, would average about 20 kg per cow per day. This intake lag should be taken into account when formulating the TMR for early postpartum cows.

For component fed herds, cows should be brought up gradually on concentrate intake over the first six weeks of lactation with intake of forages relative to concentrates monitored closely to ensure that C:F ratio does not exceed 60:40. Cows should be consuming about 5 kg of concentrate dry matter at calving and be held at that level for three to four days. Then intake of concentrate dry matter can be increased at the rate of .25 kg per day for multiparous cows and .20 kg per day for primiparous cows until peak levels of concentrate feeding are achieved during the sixth week postpartum. This feeding schedule will hold C:F ratios under 60:40 assuming normal forage intakes. Often times ruminal acidosis is caused by having cows on full concentrates during the second week postpartum resulting in C:F ratios of 70:30 or even 80:20. Slug feeding grain should be avoided. Feeding grain at least 3 to 4 times per day and offering some forage before grain can improve ruminal fiber digestion. Feeding the protein supplement when the grain is fed may improve rumen digestion by synchronizing ammonia and VFA in the rumen.

Carbohydrates

Carbohydrate nutrition is probably the most challenging aspect of feeding dairy cows, and is the area most commonly associated with laminitis. Sufficient chemical fiber is needed to maintain rumen function, prevent ruminal acidosis, and keep cows on feed. Sufficient physical fiber is needed to promote rumination activity and saliva flow, maintain adequate rumen fill, and prevent displaced abomasum. Sufficient NSC is needed to provide adequate energy to support milk production and body condition requirements. Fiber and NSC fractions must be balanced properly, particularly in early postpartum rations.

Carbohydrates comprise about 70 percent of the dry matter in typical dairy rations. Total feedstuff carbohydrate can be separated into cell wall and noncell wall components termed fiber and nonstructural carbohydrate, respectively. Neutral detergent fiber (NDF) contains lignin, cellulose and hemicellulose, the indigestible or slowly digested components of feeds. Starch, sugars and pectin make up the highly digestible carbohydrate or NSC fraction of feeds. By subtracting percent NDF, crude protein (CP), ether extract and ash from 100, we can estimate percent NSC.

Dairy cows require a minimum amount of fiber and forage in the diet for proper chewing and rumination activity, to maintain rumen pH above 6.2 and acetate:propionate ratio above 2.5, and for proper rumen function. Fine chopping reduces the "effective" fiber content of forages. Maximum restrictions on dietary NSC help prevent ruminal acidosis.

Carbohydrate status of dairy cattle diets has traditionally been evaluated with regard to measures of structural carbohydrate content; NDF or acid detergent fiber (ADF). Field

nutritionists are looking more closely at NSC. Evaluation of the carbohydrate status of milking cow rations should include:

- amounts of total ration and forage dry matter fed and consumed.
- concentrations of total ration NDF and ADF and NDF from forage.
- proportion of forage in the ration and forage particle length.
- content of total ration NSC.
- ruminal fermentability of NSC for grains and byproduct feeds.
- frequency and sequence of feeding forage and grain in component fed herds.

One of the primary benefits of TMR's is that they allow us to get a better handle on total ration and forage dry matter intake compared to systems where forage and grain are offered separately. Since nutrients are contained in the dry matter portion of feedstuffs, routine and accurate measurement of the moisture content of feeds is important so that the necessary ration adjustments can be made to keep recommended nutrient intakes on target. This is particularly important for TMR's; moisture content of forages should be monitored on-farm at least weekly as well as each time a change in forage moisture content is apparent.

Remember that on many farms there are three rations; the one formulated by the nutritionist, the one fed by the feeder, and the one actually consumed by the cow. Carbohydrate feeding guidelines must be compared with the ration that the cow consumes. When the formulated ration "looks great on paper" relative to feeding standards but ruminal acidosis is apparent, always remember to determine how much wet total ration and forage is actually being consumed, check the moisture content of wet forages, grains and byproduct feeds to recalculate dry matter intakes, check NDF and ADF analyses on forages to ensure that estimates of fiber

intakes are accurate, and check for proper feeding rates, scale calibration and feed mixing.

Minimum Fiber and Forage

When feeding high producing, early lactation cows we must meet minimum fiber needs first, energy needs second. When troubleshooting ration carbohydrates your first check should always be to ensure that minimum fiber and forage needs are being met. Compare rations to the following minimum fiber and forage guidelines (dry matter basis) for high producing cows fed haycrop forage predominantly: 19 to 21% ADF, 21% NDF from forage, 40 to 45% forage, and 27 to 30% total ration NDF. The total ration NDF content will generally be closer to 35% for rations containing barley or high-fiber byproduct based concentrates, but minimum NDF from forage content and maximum NSC content must be closely monitored.

Formulation of rations to contain at least 21% NDF from forage works well to set the minimum percent forage in the ration dry matter for haycrop forage based rations. This results in minimum percent forage in the ration dry matter ranging from 60% with low fiber (35% NDF) forage to 40% with high fiber (55% NDF) forage. Therefore, minimum amounts of forage dry matter intake range from 13.6 to 9.1 kg per cow per day to meet minimum fiber needs as we go from low to high fiber forages for cows averaging 22.7 kg of total dry matter intake. When troubleshooting ration carbohydrates the first and most important number that should be calculated is NDF from forage.

When byproduct fiber sources, such as whole cottonseed are fed, haycrop forage based rations with 18% NDF from forage should be adequate (Clark and Armentano, 1992). Rations with 50% or more well-eared corn silage in the forage dry matter should contain at least 23% NDF from

forage. Here again, inclusion of whole cottonseed in the ration may allow a slightly lower level of ration NDF from forage of 21%.

Fineness of chop can alter the effectiveness of forage fiber for maintaining chewing activity. Forage in all haycrop silage based rations should be chopped with the chopper set at a .95 cm theoretical length of cut (TLC) to provide 25% (weight basis) of the particles greater than 5 cm long. Forage particle length needs to be evaluated when troubleshooting ration carbohydrates.

Haycrop silage chopped at a .64 cm TLC to provide only about 10% of the particles greater than 5 cm long needs to be fed with at least 2.5 kg of long hay to provide adequate "effective" fiber in the ration. Haycrop silage chopped at .48 cm TLC with less than 5 percent coarse particles should be fed with 4.5 kg of long hay. The bottom forage level may need to be set at 23% NDF from forage when finely chopped haylage is fed and no long hay is available. Response to sodium bicarbonate buffers in haycrop silage based rations has been small in research trials. However, adding buffers to rations containing finely chopped silages may help if saliva production is low because of low roughage value in the ration. Holsteins need to chew 11 to 12 hours per day to maintain normal rumen function. Addition of buffers at .75% of total ration dry matter is common with corn silage based rations. Alfalfa is generally more forgiving with regard to ruminal acidosis than corn silage or other grasses, because of its higher inherent buffering capacity. Also, the pH of alfalfa silage is generally higher than corn or grass silages (4.5-5.0 vs. 3.5-4.0). High dietary acidity is also of concern when high-moisture grains are fed. Sodium bicarbonate buffer addition to high acidity rations is recommended.

Nonstructural Carbohydrate

NSC should not exceed 40-45% of ration dry matter. Check to make sure that this upper limit is not exceeded when corn silage based diets are fed, since well-eared corn silage contains about 15 percentage units more NSC than alfalfa of a similar NDF content.

The optimum ration NSC is not well defined. New York workers suggest that 40 percent NSC is desired; West Virginia workers suggest 38 to 40%. Our work suggests that 35 to 38% NFC is acceptable. Byproduct feeds tend to be higher in fiber and lower in NSC than shelled corn. These feeds can help reduce the highly digestible carbohydrate load in the cows' rumen, particularly when incorporated into corn silage based or high grain rations. This may improve ruminal pH and fiber digestion in the rumen.

When evaluating ration carbohydrates it is important to consider inherent differences between feeds in the ruminal fermentability of carbohydrates. The NDF in soy hulls and beet pulp is highly fermentable in the rumen and these feeds can be used to supply fermentable fiber in the ration as starch replacers. Inclusion of these ingredients in early postpartum TMR's allows the formulation of high NDF, moderate NSC diets of high energy density. However, they have limited value as forage replacers. The NDF in whole cottonseed is effective in partially replacing forage NDF (Clark and Armentano, 1992).

Starch in barley and wheat is degraded more rapidly in the rumen than starch in corn. Steam flaking grain increases starch digestion in the rumen. Starch in high-moisture grain is more rapidly degraded in the rumen than starch in dry grain. Starch in finely ground grain is degraded more rapidly in the rumen than starch in coarsely processed grain.

When rates of starch degradation in the rumen are too fast, acidosis can result. Caution is advised when feeding NSC sources with high ruminal degradability, such as barley or wet, finely ground high-moisture corn. It is recommended that the NSC content of rations with these highly fermentable grain sources be limited to 35%. Sodium bicarbonate buffer addition to the rations is also recommended. Avoid very wet (>35% moisture), finely ground high-moisture corn. A coarse roll is preferred for high-moisture corn. A medium to fine grind is preferred for dry corn and drier high-moisture (<25% moisture) corn, particularly when fed in a TMR.

Feeding Guidelines For Preventing Laminitis

1. Formulate rations to meet or exceed minimum National Research Council (NRC; 1989) guidelines for acid detergent fiber (ADF) and neutral detergent fiber (NDF).

2. Rations should contain a minimum of 18% to 21% NDF from forage.

3. Silages should be chopped to contain 25% of the particles (weight basis) over 5 cm long.

4. If silages are chopped too fine, consider feeding 2.5 to 4.5 kg of long or coarsely chopped hay per cow daily.

5. Do not exceed 35% to 40% NSC in the ration, depending on the grain source.

6. Control ruminal fermentability of supplemental NSC by partially substituting corn for barley or wheat, and(or) including highly digestible fiber sources (i.e. beet pulp, soy hulls, or whole cottonseed) in the concentrate.

7. Supplement dietary buffers in early lactation. The recommended feeding rate of

sodium bicarbonate is .75% of total ration dry matter.

8. Feed a TMR to control C:F ratio. Closely monitor changes in forage moisture content and adjust rations accordingly.

9. For herds feeding concentrates and forages separately: feed concentrates at least 3 to 4 times daily, monitor intake of forages relative to concentrates to ensure that C:F ratio does not exceed 60:40, and bring cows up gradually on concentrate intake over the first six weeks of lactation.

10. Provide a steam-up ration two weeks prior to calving with cows receiving concentrate up to .5% to .75% of body weight or 3.5 to 5.0 kg per cow daily.

References

Bazeley, K. and P.J.N. Pinsent. 1984. Preliminary observations on a series of outbreaks of acute laminitis in cattle. Vet. Rec. 115:619.

Clark, P.W. and L.E. Armentano. 1992. The effectiveness of NDF in cottonseed and dried distiller's grains. J. Dairy Sci. 75(Suppl.1):233(Abstr.).

Dirksen, G.U., H.G. Liebich and E. Mayer. 1985. Adaptive changes of the ruminal mucosa and their functional and clinical significance. Bov. Pract. Nov., 1985. pp. 116-120.

Kertz, A.F., L.F. Reutzel and G.M. Thomson. 1991. Dry matter intake from parturition to midlactation. J. Dairy Sci. 74:2290.

Livesey, C.T. and F.L. Fleming. 1984. Nutritional influences on laminitis, sole ulcer, and bruised sole in Friesian cows. Vet. Rec. 114:510 - 512.

Manson, F.J. and J.D. Leaver. 1989. The effect of concentrate : silage ratio and of hoof trimming on lameness in dairy cattle. Anim. Prod. 49:15 - 22.

Manson, F.J. and J.D. Leaver. 1988. The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. 47:185 - 190.

National Research Council. 1989. Nutrient requirements of dairy cattle. 6th Rev. Ed. Nat. Acad. Press. Washington, D.C.

Nocek, J.E. 1985. Management of foot and leg problems in dairy cattle. The Prof. Anim. Sci. 1:1-7.

Pehrson, B.G. and R.D. Shaver. 1992. Displaced abomasum: Clinical data and effects of peripartal feeding and management on incidence. Proc. AABP World Buiatrics Congress. St. Paul, MN.

Shaver, R.D. 1993. TMR strategies for transition feeding of dairy cows. Proc. MN Nutr. Conf. Bloomington, MN.

Van Saun, R.J. 1992. Nutritional management of the dry cow. Proc. MI Dairy Mgmt. Conf. Grand Rapids, MI.

HISTOPATHOLOGICAL FINDINGS OF THE DIGITS IN DAIRY COWS IN JAPAN

Manabu Mochizuki and Nobuo Sasaki

Department of Veterinary Surgery
Faculty of Agriculture
The University of Tokyo
1-1-1 Yayoi, Bunkyo-ku
Tokyo 113, Japan

Two hundred thirty-nine digits of 45 Holstein dairy cows, which were raised in typical Japanese dairy farms and received poor hoof management, were randomly obtained in the slaughterhouses and examined histopathologically. The findings were classified into 5 grades by the severity of circulatory disturbances and of keratogenesis. The lesions from Grade 1 to 5 were considered as manifestations of serial lesions indicating that subclinical laminitis advanced to other hoof lesions. The incidence of Grade 2, regarded as subclinical laminitis, reached approximately 50% of digits examined. The lesions classified as Grade 3 (23.9%) and 4 (5.4%) were mainly characterized by circulatory disturbances, which were similar to those of chronic laminitis in previous reports. The incidence of Grade 5, characterized by sole ulcer, was 5.4%. It is suggested that a considerable number of dairy cows in Japan suffer from subclinical laminitis, which may be the cause of recent high incidence of hoof diseases in dairy cows.

A LIGHT & SCANNING ELECTRON MICROSCOPIC STUDY OF THE EPIDERMIS IN THE REGION OF A SOLE ULCER IN A DAIRY COW

K.A. Leach[1], *S.A. Kempson*[1], *and D.N. Logue*[2]

[1] Royal (Dick) School of Vet Studies
Summerhall, Edinburgh EH9 1QH, Scotland

[2] The Scottish Agricultural College
Auchincruive, Ayr KA6 5HW, Scotland

A dairy cow, six years old, was culled because of severe lameness caused by persistent sole ulcers on both outer hind claws. The hind feet were prepared for light and scanning electron microscopy. The centre of the ulcer cavity was filled with a plug of white granulation tissue. The central region of the ulcer showed no evidence of epidermal tissue. Surrounding the ulcer site, there were changes in the composition of the corium, the structure and thickness of the epidermis, and the appearance of horn. As histological changes were restructured to the immediate region of the ulcer, it is thought that the original insult was also localised. Alternate regions of normal and abnormal horn had formed in response to the trauma. Thus the tissue responses designed to restore normal structure and function had not been wholly successful.

TREATMENT OF SERIOUS CLAW DISEASES AT THE CLINIC FOR RUMINANTS, LJUBLJANA, SLOVENIA

T. Zadnik, T. Modic, and I. Jazbec

Veterinary Faculty
Cesta v Mestni log 47
61000 Ljubljana, Slovenia

At our clinic in Ljubljana we have performed several demanding claw surgical interventions in recent years. In 32 cases we have found solear ulceration s.c. Rusterholz ulcer. The disease affected mostly older dairy cows and one of the factors that strongly influenced the seriousness of the disease was the intensive dairy husbandry with prolonged housing periods. Nine claw amputations were carried out as well. The so-called "high amputation" proved a very successful procedure of treatment. The surgery of the affected digit is rather simple and satisfactory. The animals are fully recovered 5 to 8 weeks after intervention.

MICROVASCULARIZATION OF THE CATTLE DIGIT

Tetsuo Nasu, Kouhei Kitazaki[1], Masaaki Nakai, and Hiroyuki Ogawa[1]

[1] Departments of Veterinary Anatomy and Surgery
Faculty of Agriculture
Miyazaki University
Miyazaki, Japan 889-21

The blood supply of the digits of six young cattle's forelimbs were studied using a microvascular casting corrosion technique and scanning microscopy. The histological observations by the light microscopy of the sections of the decalcificated digit were added. We observed the vessels of the dermal lamina, dermal papilla and transitional part between them. In the laminar region of the claw the capillary consisted of thin parallel sheets arranged in vertical rows. Each sheet consisted of many vessels that coursed to the surface of the claw vertically. These vessels oriented parallel to one another and anastomosed like a ladder. This ladder structure of the vessels was surrounded by smaller calibre interconnecting vessels. In the papilla regions the shape of the vascular cast was a convex brush border made up of numerous tapering cone-shaped papillae. The conical cast of each papillary was made up of two central vessels which ran through the entire length of the papilla. They were branched and anastomosed to make a capillary plexus. There were vascular loops at the apices of the cones. The transitional parts were recognized between the sole and abaxial wall of the claw. In these parts the vascular casts made the double flat cones that anastomosed each other. The basal portion of these structures formed continuous parallel sheets same as those in the dermal lamellae. From the histological observation, the vascular structure in all these dermal parts showed a typical capillary type that was made of one or two endothelial cells and pericytes. No arteriovenous anastomoses was observed from the cast.